www.rcjournal.com

AUGUST 1999
VOLUME 44
NUMBER 8
ISSN 0020-1324-RECACP

RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
44TH YEAR—ESTABLISHED 1956

AARC

45th International Respiratory Congress
December 13-16 • Las Vegas, Nevada

EDITORIALS

Respiratory Therapists and Conscious Sedation

ORIGINAL CONTRIBUTIONS

Comparison of Conventional Heated Humidification with a New Active Hygroscopic Heat and Moisture Exchanger in Mechanically Ventilated Patients

Effects of Tracheal Gas Insufflation and Tracheal Gas Exsufflation on IPEEP and CO_2 Elimination

Comparison of Demand Oxygen Delivery Systems and Continuous Flow Oxygen

Clinical Validation of the Functional Performance Inventory in Patients with COPD

REVIEWS, OVERVIEWS, & UPDATES

Dry Powder Inhalers

SPECIAL ARTICLES

Bicarbonate, Oh, Bicarbonate

CRCE THROUGH THE JOURNAL

LEADING INSTITUTIONS HAVE RELIED ON THE ACCURACY OF ASTECH INSTRUMENTS FOR YEARS.*

Just as ancient institutions relied on the Aztec calendar for vital measurements of time, today's leading medical institutions rely on ASTECH Peak Flow Meters for vital patient measurements.* Because more accurate information leads to more effective therapy.

Entrust the health of your patients to the peak flow meter trusted by some of the leading medical hospitals for pulmonary medicine.

The ASTECH Peak Flow Meter. A precision instrument with a record of accuracy and reproducibility that has stood the test of time in the most demanding environments.

Call 800-527-4278 today for really accurate information about a really accurate peak flow meter.

ASTECH® PEAK FLOW METER

DEY

©1999 DEY. All rights reserved.
09-733-00 1/99

AARC ZENITH AWARD '97 & '98

* National Jewish Medical and Research Center (ranked #1 for pulmonary diseases by *U.S. News and World Report*, July 1998) and other thought leaders use the Astech Peak Flow Meter exclusively.

Circle 133 on reader service card

Ipratropium Bromide

INHALATION SOLUTION

From DEY

The only generic alternative to Atrovent® with Patient-Focused Packaging.™

Plastic unit-dose vials with DEY TwistFlex™ tops provide easy access just when it's needed most.

Color-coded packages and vial labels provide quick identification.

AN-rated DEY Ipratropium Bromide is the high-quality, low-cost* generic alternative to Atrovent® Inhalation Solution. It assures quick, convenient dispensing, reducing the chance of patient errors. And now, it is available in 25-, 30-, and 60-vial packs.

For more information about Ipratropium Bromide Inhalation Solution from DEY Laboratories, contact your DEY representative or call **1-800-755-5560.**

Now available in 25s, 30s, and 60s!

*Prices are based on the current published average wholesale prices for Atrovent® Inhalation Solution and DEY's Ipratropium Bromide Inhalation Solution.

Atrovent® is a registered trademark of Boehringer Ingelheim Pharmaceuticals, Inc.

© 1997 DEY Laboratories
09-720-01 09/97

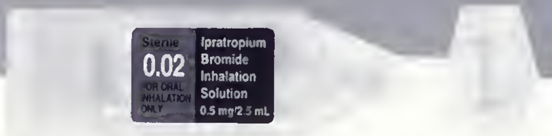
DEY Breathing New Life Into Respiratory Care

DEY Laboratories
2751 Napa Valley Corporate Drive
Napa, CA 94558

(Please see adjacent page for summary of prescribing information)

Circle 114 on reader service card

Patient-Focused Packaging™



Now available in 25s, 30s, and 60s!

Ipratropium Bromide

INHALATION SOLUTION

From DEY

Ipratropium Bromide Inhalation Solution 0.02%

BRIEF SUMMARY: Please see package insert for full prescribing information.

CONTRAINDICATIONS: Ipratropium bromide is contraindicated in known or suspected cases of hypersensitivity to ipratropium bromide, or to atropine and its derivatives.

WARNINGS: The use of Ipratropium Bromide Inhalation Solution as a single agent for the relief of bronchospasm in acute COPD exacerbation has not been adequately studied. Drugs with faster onset of action may be preferable as initial therapy in this situation. Combination of Ipratropium Bromide Inhalation Solution and beta agonists has not been shown to be more effective than either drug alone in reversing the bronchospasm associated with acute COPD exacerbation.

Immediate hypersensitivity reactions may occur after administration of ipratropium bromide, as demonstrated by rare cases of urticaria, angioedema, rash, bronchospasm and oropharyngeal edema.

PRECAUTIONS: General: Ipratropium bromide should be used with caution in patients with narrow angle glaucoma, prostatic hypertrophy or bladder neck obstruction.

Information for Patients: Patients should be advised that temporary blurring of vision, precipitation or worsening of narrow-angle glaucoma or eye pain may result if the solution comes into direct contact with the eyes. Use of a nebulizer with mouthpiece rather than face mask may be preferable, to reduce the likelihood of the nebulizer solution reaching the eyes. Patients should be advised that Ipratropium Bromide Inhalation Solution can be mixed in the nebulizer with albuterol or metaproterenol if used within one hour. Drug stability and safety of Ipratropium Bromide Inhalation Solution when mixed with other drugs in a nebulizer have not been established. Patients should be reminded that Ipratropium Bromide Inhalation Solution should be used consistently as prescribed throughout the course of therapy.

Drug Interactions: Ipratropium bromide has been shown to be a safe and effective bronchodilator when used in conjunction with beta adrenergic bronchodilators. Ipratropium bromide has also been used with other pulmonary medications, including methylxanthines and corticosteroids, without adverse drug interactions.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Two-year oral carcinogenicity studies in rats and mice have revealed no carcinogenic potential at dietary doses up to 6 mg/kg/day of ipratropium bromide.

Results of various mutagenicity studies (Ames test, mouse dominal lethal test, mouse micronucleus test and chromosome aberration of bone marrow in Chinese hamsters) were negative.

Fertility of male or female rats at oral doses up to 50 mg/kg/day was unaffected by ipratropium bromide administration. At doses above 90 mg/kg, increased resorption and decreased conception rates were observed.

Pregnancy: *TERATOGENIC EFFECTS:*
Pregnancy Category B. Oral reproduction studies performed in mice, rats and rabbits at doses of 10, 100, and 125 mg/kg respectively and inhalation reproduction studies in rats and rabbits at doses of 1.5 and 1.8 mg/kg (or approximately 38 and 45 times the recommended human daily dose) respectively, have demonstrated no evidence of teratogenic effects as a result of ipratropium bromide. However, no adequate or well controlled studies have been conducted in pregnant women. Because animal reproduction studies are not always predictive of human response, ipratropium bromide should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether ipratropium bromide is excreted in human milk. Although lipid-insoluble quaternary bases pass into breast milk, it is unlikely that ipratropium bromide would reach the infant to a significant extent, especially when taken by inhalation since ipratropium bromide is not well absorbed systemically after inhalation or oral administration. However, because many drugs are excreted in human milk, caution should be exercised when ipratropium bromide is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in pediatric patients below the age of 12 have not been established.

ADVERSE REACTIONS: Adverse reaction information concerning Ipratropium Bromide Inhalation Solution is derived from 12-week active-controlled clinical trials. Additional information is derived from foreign post-marketing experience and the published literature.

All adverse events, regardless of drug relationship, reported by three percent or more patients in the 12-week controlled clinical trials appear in the table.

All Adverse Events, From a Double-blind, Parallel, 12-week Study of Patients with COPD*

	PERCENT OF PATIENTS				
	Ipratropium Bromide (500 mcg t.i.d.) n = 219	Meta-proterenol (15 mg t.i.d.) n = 212	Ipratropium / Bromide/ Metaproterenol (500 mcg t.i.d./ 15 mg t.i.d.) n = 108	Albuterol (2.5 mg t.i.d.) n = 205	Ipratropium Bromide/ Albuterol (500 mcg t.i.d 2.5 mg t.i.d.) n = 100
Body as a Whole-General Disorders					
Headache	6.4	5.2	6.5	6.3	9.0
Pain	4.1	3.3	0.9	2.9	5.0
Influenza-like symptoms	3.7	4.7	6.5	0.5	1.0
Back Pain	3.2	1.9	1.9	2.4	0.0
Chest Pain	3.2	4.2	5.6	2.0	1.0
Cardiovascular Disorders					
Hypertension/ Hypertension Aggravated	0.9	1.9	0.9	1.5	4.0
Central & Peripheral Nervous System					
Dizziness	2.3	3.3	1.9	3.9	4.0
Insomnia	0.9	0.5	4.6	1.0	1.0
Tremor	0.9	7.1	8.3	1.0	0.0
Nervousness	0.5	4.7	6.5	1.0	1.0
Gastrointestinal System Disorders					
Mouth Dryness	3.2	0.0	1.9	2.0	3.0
Nausea	4.1	3.8	1.9	2.9	2.0
Constipation	0.9	0.0	3.7	1.0	1.0
Musculo-skeletal System Disorders					
Arthritis	0.9	1.4	0.9	0.5	3.0
Respiratory System Disorders (Lower)					
Coughing	4.6	8.0	6.5	5.4	6.0
Dyspnea	9.6	13.2	16.7	12.7	9.0
Bronchitis	14.6	24.5	15.7	16.6	20.0
Bronchospasm	2.3	2.8	4.6	5.4	5.0
Sputum Increased	1.4	1.4	4.6	3.4	0.0
Respiratory Disorder	0.0	6.1	6.5	2.0	4.0
Respiratory System Disorders (Upper)					
Upper Respiratory Tract Infection	13.2	11.3	9.3	12.2	16.0
Pharyngitis	3.7	4.2	5.6	2.9	4.0
Rhinitis	2.3	4.2	1.9	2.4	0.0
Sinusitis	2.3	2.8	0.9	5.4	4.0

*All adverse events, regardless of drug relationship, reported by the three percent or more patients in the 12-wee controlled clinical trials

Additional adverse reactions reported in less than three percent of the patients treated with ipratropium bromide include tachycardia, palpitations, eye pain, urinary retention, urinary tract infectio and urticaria. Cases of precipitation or worsening of narrow-angle glaucoma and acute eye pain hav been reported.

Lower respiratory adverse reactions (bronchitis, dyspnea and bronchospasm) were the most commo events leading to discontinuation of ipratropium bromide therapy in the 12-week trials. Headach mouth dryness and aggravation of COPD symptoms are more common when the total daily dose o ipratropium bromide equals or exceeds 2,000 mcg.

Allergic-type reactions such as skin rash, angioedema of tongue, lips and face, urticaria, laryr gospasm and anaphylactic reaction have been reported. Many of the patients had a history o allergies to other drugs and/or foods.

HOW SUPPLIED: Ipratropium Bromide Inhalation Solution is a clear, colorless solution supplied in a unit dose vial containing 2.5 mL. Supplied in cartons as listed below:

25 vials per carton	NDC 49502-685-03
30 vials per carton	NDC 49502-685-33
60 vials per carton	NDC 49502-685-60

Each vial is made from a low density polyethylene (LDPE) resin.

Store between 15° and 30°C (59° and 86°F). Protect from light. Retain in carton until time of use

Caution: Federal law prohibits dispensing without prescription.

DEY

DEY Laboratories, Napa, CA 94558
03-343-02(BRS) April 1997

FOR INFORMATION, CONTACT:

AARC Membership or Other AARC Services
American Association for Respiratory Care
11030 Ables Ln
Dallas TX 75229-4593
(972) 243-2272 • Fax (972) 484-2720
http://www.aarc.org

Therapist Registration or Technician Certification
National Board for Respiratory Care
8310 Nieman Rd
Lenexa KS 66214
(913) 599-4200 • Fax (913) 541-0156
http://www.nbrc.org

Accreditation of Education Programs
Committee on Accreditation for Respiratory Care
1701 W Euless Blvd, Suite 300
Euless TX 76040
(817) 283-2835 • Fax (817) 354-8519
http://www.coarc.com

Grants, Scholarships, Community Projects
American Respiratory Care Foundation
11030 Ables Ln
Dallas TX 75229-4593
(972) 243-2272 • Fax (972) 484-2720

Government Affairs —
Cheryl West MHA (703-548-8506)

State Government Affairs —
Jill Eicher MPA (703-548-8538)
1225 King St, Second Floor
Alexandria VA 22314
Fax (703) 548-8499

RESPIRATORY CARE

RESPIRATORY CARE (ISSN 0020-1324, USPS 0489-190) is published monthly by Daedalus Enterprises Inc, at 11030 Ables Lane, Dallas TX 75229-4593, for the American Association for Respiratory Care. One volume is published per year beginning each January. Subscription rates are $75 per year in the US; $90 in all other countries (for airmail, add $94).

The contents of the Journal are indexed in Hospital and Health Administration Index, Cumulative Index to Nursing and Allied Health Literature, EMBASE/Exerpta Medica, and RNdex Library Edition. Abridged versions of RESPIRATORY CARE are also published in Italian, French, and Japanese, with permission from Daedalus Enterprises Inc.

Periodicals postage paid at Dallas TX and at additional mailing offices. POSTMASTER: Send address changes to RESPIRATORY CARE, Membership Office, Daedalus Enterprises Inc, 11030 Ables Lane, Dallas TX 75229-4593.

Printed in the United States of America
Copyright © 1999, by Daedalus Enterprises Inc.

CONTENTS

AUGUST 1999 / VOLUME 44 / NUMBER 8

EDITORIALS

Respiratory Therapists and Conscious Sedation
by Charles G Durbin Jr—Charlottesville, Virginia 909

ORIGINAL CONTRIBUTIONS

Comparison of Conventional Heated Humidification with a New Active Hygroscopic Heat and Moisture Exchanger in Mechanically Ventilated Patients
by Richard D Branson, Robert S Campbell, Jay A Johannigman, Michael Ottaway, Kenneth Davis Jr, Fred A Luchette, and Scott Frame—Cincinnati, Ohio 912

Effects of Tracheal Gas Insufflation and Tracheal Gas Exsufflation on Intrinsic Positive End-Expiratory Pressure and Carbon Dioxide Elimination
by Toshimichi Takahashi—Nagoya, Japan, and Guillermo Bugedo, Alexander B Adams, Peter L Bliss, and John J Marini—St Paul, Minnesota 918

A Bench Study Comparison of Demand Oxygen Delivery Systems and Continuous Flow Oxygen
by Peter L Bliss, Robert W McCoy, and Alexander B Adams—St Paul, Minnesota 925

Clinical Validation of the Functional Performance Inventory in Patients with Chronic Obstructive Pulmonary Disease
by Nancy Kline Leidy and Ann R Knebel—Bethesda, Maryland 932

REVIEWS, OVERVIEWS, & UPDATES

Dry Powder Inhalers
by Rajiv Dhand and James Fink—Hines, Illinois 940

SPECIAL ARTICLES

Bicarbonate, Oh, Bicarbonate
by H Frederic Helmholz Jr—Rochester, Minnesota 952

PFT CORNER

Flow-Volume Loop Analysis of Airflow Limitation: “All that Obstructs Is Not Bronchial Asthma”
by Deborah A Hunton and David A Kaminsky—Burlington, Vermont 955

PFT NUGGETS

An Obese Patient Referred for Preoperative Pulmonary Clearance
by Salim Kathawalla—Minneapolis, Minnesota, and Muzaffar Ahmad—Cleveland, Ohio 959

A 38-Year-Old Man with Progressive Dyspnea and a Clear Chest Roentgenogram
by Robert J Schilz—Cleveland, Ohio 961

LETTERS TO THE EDITOR

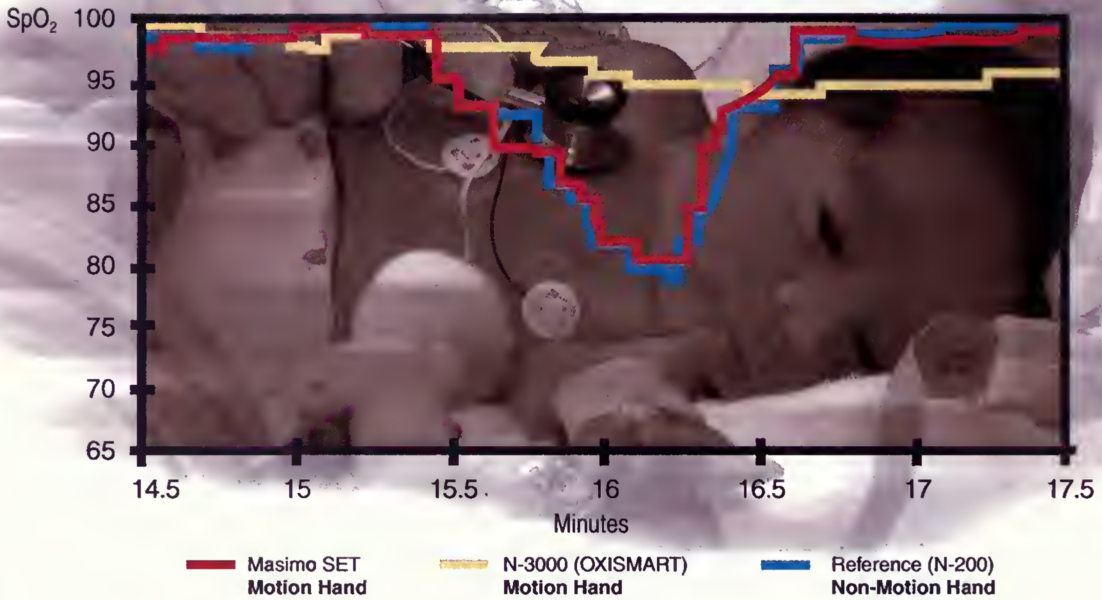
Role of Bicarbonate Ion Concentration in Acid-Base Balance
by Bob Demers—Carmel, California
response by Joe Morfei—Syracuse, New York 963

Saturation Recorded During Rubbing Motion

Masimo SET accurately tracks the desaturation and resaturation, while the Oxismart product misses the hypoxemic event.

This study was performed on adult volunteers.

Data courtesy of Dr. Steven Barker Ph.D., M.D., Professor and Chairman, Department of Anesthesiology, University of Arizona



the proof is in the performance

motion study

SpO$_2$ System	Masimo SET	OXISMART N-3000	N-200
False Alarms	0%	36%	30%
True Hypoxemias Missed	0%	16%	1%

Source: Barker SJ, Shah NK. The Effects of Motion on the Performance of Pulse Oximeters in Volunteers. *Anesthesiology* 1997;86(1):101-108.

low perfusion study

SpO$_2$ System	Masimo SET	OXISMART N-3000	N-200
Drop Out Rate	0.8%	9.9%	17.8%
True Hypoxemias Missed	0%	33.3%	33.3%

Source: Barker SJ, Novak S, Morgan S. The Performance of Three Pulse Oximeters During Low Perfusion in Volunteers. *Anesthesiology* 1997;87(3A):A409.

The facts about Masimo SET pulse oximetry:

- The only pulse oximetry technology clinically proven accurate during motion and low perfusion
- Virtually eliminates false alarms
- More reliable in detecting true alarms
- Continuous monitoring (no freezing)

Over the past two years, approximately one-third of the world's pulse oximetry suppliers have adopted Masimo SET as their pulse oximetry solution. Now, more than twenty patient monitors are available from leading patient monitoring companies with Masimo SET pulse oximetry.

Prior to the introduction of Masimo SET, conventional pulse oximeters could only be relied upon for accurate measurement during ideal conditions. Masimo SET reliably and continuously tracks changes in saturation and pulse rate, giving you accurate monitoring when you need it most, even when your patients are moving or have low perfusion.

Prove it to yourself — take Masimo SET pulse oximetry to your toughest patients and perform a confirming CO-Oximeter ABG. If the Masimo SET pulse oximeter measurement is further from the CO-Oximeter measurement than any other non-Masimo SET pulse oximeter, we will reimburse the cost of your test.*

for more information on Masimo SET pulse oximetry or to schedule a free trial evaluation, contact us by phone or internet

1-877-4-MASIMO • www.masimo.com

Circle 106 on reader service card

Masimo SET.

Instruments and sensors containing Masimo SET technology are identified with the Masimo SET logo.
Look for the Masimo SET designation to ensure accurate pulse oximetry when needed most.

* CO-Oximeter test must be performed on a calibrated CO-Oximeter with four or more wavelengths (e.g., IL 482 or Radiometer OSM3). Certain other rules and restrictions apply. Contact Masimo for more details.

© 1999 Masimo Corporation. Masimo, SET and $ are federally registered trademarks of Masimo Corporation. N-3000, OXISMART and N-200 are trademarks of Nellcor Puritan Bennett.

CONTENTS

CONTINUED...

BOOKS, FILMS, TAPES, & SOFTWARE

Understanding Acid-Base
reviewed by H Frederic Helmholz Jr—Rochester, Minnesota 965

Manual of Pulmonary Function Testing, 7th Edition
reviewed by Robert R Fluck Jr—Syracuse, New York 965

Exam Review and Study Guide for Perinatal/Pediatric Respiratory Care 967
reviewed by Robert Lewis—Seattle, Washington

Management Skills for the New Health Care Supervisor, 3rd Edition 968
reviewed by Carol L Kasper—Seattle, Washington

High Life: A History of High-Altitude Physiology and Medicine
reviewed by Colin K Grissom—Salt Lake City, Utah 968

CONTINUING EDUCATION EXAMINATION

CRCE Through the Journal – 1999 970

ALSO IN THIS ISSUE

983 AARC Membership Application

888 Abstracts from Other Journals

992 Advertisers Index & Help Lines

992 Author Index

989 Calendar of Events

985 Manuscript Preparation Guide

981 MedWatch

980 New Products & Services

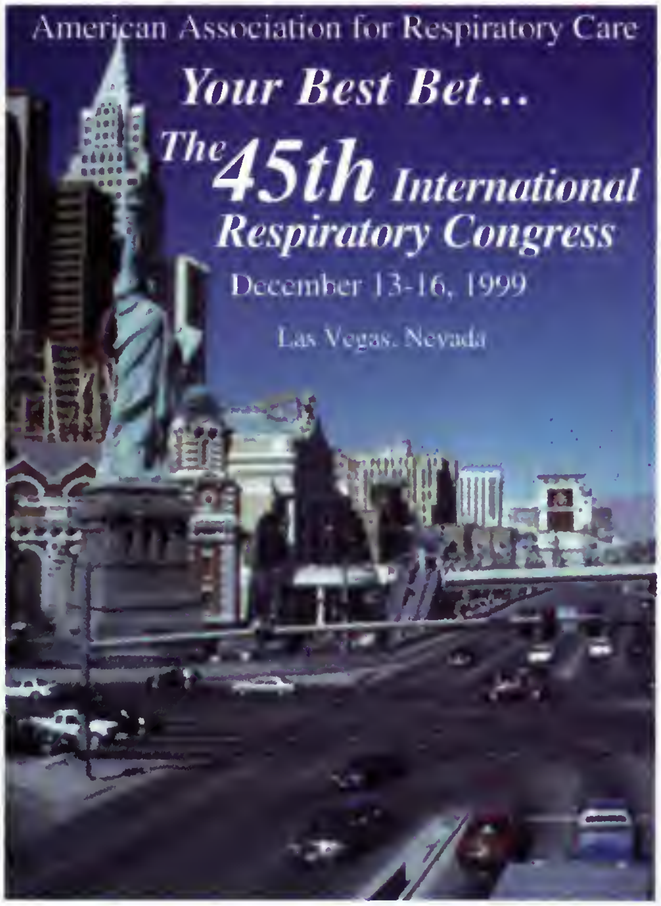
990 Notices

RESPIRATORY CARE

A Monthly Science Journal
Established in 1956

The Official Journal of the
American Association for
Respiratory Care

AARC


American Association for Respiratory Care
Your Best Bet...
The 45th International Respiratory Congress
December 13-16, 1999
Las Vegas, Nevada

Westmed provides superior aerosol therapy across the care continuum

MDILog™

Electronic monitors for aerosol delivery devices

The device provides the ability to monitor compliance and record true delivery and evaluate patient technique. These features make it the ideal disease management tool for asthma and COPD patients.

- Records date and time of each use and evaluates technique
- Reduces lengths of stay and increases staff productivity
- Transmits data for analysis/storage with fast wireless communications
- Ideal for use in acute, subacute, pulmonary rehab, physician's office and home settings

Circulaire™

Maximizing delivery of inhaled medications to the lungs

Designed for inpatient and at–home use, the Circulaire's patented distensible drug reservoir minimizes waste while it's variable resistor tailors treatment to individual pediatric/ adult patient needs.

- Optimum aerosol particle size for superior lung deposition
- Ideal for protocol-based concentrated drug delivery
- Virtual elimination of systemic reaction to beta stimulators
- "Biofeedback" gauges encourage maximum patient effort

HEART™ Nebulizers

Better outcomes at lower cost through continuous nebulization therapy

The high–output extended aerosol respiratory therapy {HEART®} system is unsurpassed in delivering continuous nebulization therapy [CNT] in inpatient and outpatient settings.

- The HEART® high–output nebulizer — up to 8 hours of therapy
- The MiniHEART® low–flow nebulizer — up to 10 hours of treatment
- The low–cost and low-flow UniHEART™ nebulizer — ideal for Emergency Department use

Westmed

A leader in aerosol drug delivery and drug management,
providing superior results through productivity gains and patient outcomes
across the care continuum.

Circle 104 on reader service card

Westmed, Inc. 3351 E. Hemisphere Loop, Tucson, Arizona 85706 · Phone: 800-724-2328 Fax: 520-294-6061 www.lungdepot.com

Label PN 70468, Rev 02

EDITORIAL OFFICE

600 Ninth Avenue, Suite 702
Seattle WA 98104
(206) 223-0558
Fax (206) 223-0563
www.rcjournal.com

MANAGING EDITOR

Ray Masferrer RRT

ASSISTANT EDITOR

Katherine Kreilkamp

EDITORIAL ASSISTANT

Linda Barcus

COPY EDITOR

Matthew Mero

PRODUCTION

Kelly Piotrowski

PUBLISHER

Sam P Giordano MBA RRT

MARKETING

Rick Owen
Director of Marketing
Tim Goldsbury
Director, Advertising Sales
Beth Binkley
Advertising Assistant

RESPIRATORY CARE

A Monthly Science Journal
Established in 1956

The Official Journal of the
American Association for
Respiratory Care

AARC

EDITOR IN CHIEF

David J Pierson MD
Harborview Medical Center
University of Washington
Seattle, Washington

ASSOCIATE EDITORS

Richard D Branson RRT
University of Cincinnati
Cincinnati, Ohio

Charles G Durbin Jr MD
University of Virginia
Charlottesville, Virginia

Dean R Hess PhD RRT FAARC
Massachusetts General Hospital
Harvard University
Boston, Massachusetts

James K Stoller MD
The Cleveland Clinic Foundation
Cleveland, Ohio

EDITORIAL BOARD

Thomas A Barnes EdD RRT
Northeastern University
Boston, Massachusetts

Michael J Bishop MD
University of Washington
Seattle, Washington

Bartolome R Celli MD
Tufts University
Boston, Massachusetts

Robert L Chatburn RRT FAARC
University Hospitals of Cleveland
Case Western Reserve University
Cleveland, Ohio

Luciano Gattinoni MD
University of Milan
Milan, Italy

John E Heffner MD
Medical University of South Carolina
Charleston, South Carolina

Mark J Heulitt MD
University of Arkansas
Little Rock, Arkansas

Leonard D Hudson MD
University of Washington
Seattle, Washington

Robert M Kacmarek PhD RRT FAARC
Massachusetts General Hospital
Harvard University
Boston, Massachusetts

Toshihiko Koga MD
Koga Hospital
Kurume, Japan

Marin H Kollef MD
Washington University
St Louis, Missouri

Patrick Leger MD
Clinique Médicale Edouard Rist
Paris, France

Neil R MacIntyre MD FAARC
Duke University
Durham, North Carolina

John J Marini MD
University of Minnesota
St Paul, Minnesota

Shelley C Mishoe PhD RRT FAARC
Medical College of Georgia
Augusta, Georgia

Joseph L Rau PhD RRT
Georgia State University
Atlanta, Georgia

Catherine SH Sassoon MD
University of California Irvine
Long Beach, California

Arthur S Slutsky MD
University of Toronto
Toronto, Ontario, Canada

Martin J Tobin MD
Loyola University
Maywood, Illinois

STATISTICAL CONSULTANT

Gordon D Rubenfeld MD
University of Washington
Seattle, Washington

SECTION EDITORS

Hugh S Mathewson MD
Joseph L Rau PhD RRT
Drug Capsule

Charles G Irvin PhD
Gregg L Ruppel MEd RRT RPFT FAARC
PFT Corner

Richard D Branson RRT
Robert S Campbell RRT
Kittredge's Corner

Jon Nilsestuen PhD RRT FAARC
Ken Hargett RRT
Graphics Corner

Patricia Ann Doorley MS RRT
Charles G Durbin Jr MD
Test Your Radiologic Skill

Editorials, Commentaries, and Reviews to Note

Pneumothorax and Systemic Air Embolism During Positive-Pressure Ventilation—Ibrahim AE, Stanwood PL, Freund PR. Anesthesiology 1999 May;90(5):1479-1481.

Laryngeal Granuloma Caused By Short-Term Endotracheal Intubation—Kaneda N, Goto R, Ishijima S, Kawakami S, Park K, Shima Y. Anesthesiology 1999 May;90(5):1482-1483.

Ventilation Difficulties Through the Intubating Laryngeal Mask—Seto A, Aoyama K, Takenaka I, Kadoya T. Anesth Analg 1999 May;88(5):1181-1182.

Neonatal Respiratory Care and Infant Mortality in Emerging Countries (editorial)—Sola A, Farina D. Pediatr Pulmonol 1999 May;27(5):303-304.

Liquid Ventilation—Melo J, Anzueto A. Intensive Care Med 1999 Apr;25(4):420-421.

International Aspects of US Government Tobacco Bills—Mackay J. JAMA 1999 May 19; 281(19):1849-1850.

Acute Respiratory Distress Syndrome: A Clinical Update—Soubani AO, Pieroni R. South Med J 1999 May;92(5):450-457.

Nitric Oxide in Adult Lung Disease—Hart CM. Chest 1999 May;115(5):1407-1417.

Postoperative Ventilatory Management—Price JA, Rizk NW. Chest 1999 May;115(5 Suppl): 130S-137S.

Assessing and Modifying the Risk of Postoperative Pulmonary Complications—Doyle RL. Chest 1999 May;115(5 Suppl):77S-81S.

Preoperative Assessment of Pulmonary Risk—Ferguson MK. Chest 1999 May;115(5 Suppl): 58S-63S.

Significant Tracheal Obstruction Causing Failure to Wean in Patients Requiring Prolonged Mechanical Ventilation: A Forgotten Complication of Long-Term Mechanical Ventilation—Rumbak MJ, Walsh FW, Anderson WM, Rolfe MW, Solomon DA. Chest 1999 Apr;115(4):1092–1095.

INTRODUCTION: Modern low-pressure, high-volume cuffed tracheotomy tubes have been shown to decrease tracheal injury. However, injury still occurs in patients requiring prolonged mechanical ventilation and prevents weaning, delays decannulation, prolongs hospitalization, and may totally obstruct the airway. We describe 37 patients, including the first reported case of failure to wean due to tracheal obstruction. METHODS: Over a 3-year period, from September 1994 to August 1997, the hospital records of 37 patients requiring prolonged mechanical ventilation (> 4 weeks) and found to have tracheal obstruction were reviewed retrospectively. They were a subgroup of 756 patients admitted to hospitals during the same period. The average endotracheal/tracheostomy cannulation time was 3 weeks/12 weeks (range 2 to 4 weeks/8 to 14 weeks). Average age was 76 years (range, 34 to 81). Underlying diseases included COPD, postcoronary artery bypass graft surgery, postpneumonectomy, severe pneumonia, acute lung injury, and ischemic heart disease. RESULTS: All 37 patients who initially failed to wean had difficulty in breathing and developed intermittent high peak airway pressures either early or during the weaning process or just on being ventilated. The insertion of a longer tracheal tube bypassed the obstruction, reestablished the airway, decreased peak airway pressures, and allowed the patient to breathe more easily. The obstruction was confirmed on bronchoscopy. Treatment consisted of either placement of a longer tracheal tube (34 of 37 patients) or placement of a tracheal stent. All but two of the patients (5.4%) were able to be weaned within a week. The two patients who still failed to be weaned were subsequently diagnosed as having amyotrophic lateral sclerosis. CONCLUSION: Tracheal obstruction in patients requiring prolonged mechanical ventilation prevented weaning. Reestablishment of the airway with a longer tracheal tube or tracheal stent allowed most of the patients to be weaned.

The Unidirectional Valve Is the Best Method to Determine Maximal Inspiratory Pressure During Weaning—Caruso P, Friedrich C, Denari SD, Ruiz SA, Deheinzelin D. Chest 1999 Apr;115(4):1096–1101.

If you've never acted on ImPulse before,

there really is no time like the present.

...for those on oxygen who want to feel capricious, fully mobile, or even *glide like the wind*...

- ImPulse™ Select provides lightweight portability for oxygen patients "on the go"
- Designed to switch between two modes of operation:
  - Pulse/intermittent breaths (average 6:1 conservation rate)
  - Pulse/every breath (average 3:1 conservation rate)
- Pulse modes offer up to 6 lpm equivalency
- Patient-selectable continuous flow feature
- Uses one 'D' size battery for greatest ambulation time; also optional power supply
- Audible and visual alarms ensure safe, reliable operation
- System securely and easily fits over top of cylinder for greater patient convenience

AirSep's New ImPulse™ Select
Oxygen Conserving Device

Act on ImPulse!

Contact AirSep to find out more:
USA/Canada Toll-Free: 800-874-0202
Tel: (716) 691-0202
Fax: (716) 691-4141
E-mail: marketing@airsep.com
Internet: www.airsep.com

NAMES
AirSep joins NAMES in support of the industry and you

© 1998 AirSep Corporation.

Circle 140 on reader service card

OBJECTIVES: Although maximal inspiratory pressure (MIP) is used as an index of inspiratory muscular strength, there is no consensus on how to measure it. We compared, during weaning from mechanical ventilation, two methods of measurement to determine which shows the greater values (MIPbest) and is more reproducible. One method measured MIP when negative pressure was maintained for at least 1 s after a forceful expiration, and the other method measured MIP with a unidirectional expiratory valve (MIPuni). DESIGN: The study had a crossover design, and patients randomly performed three measurements of each method (t1). The procedure was repeated by the same observer after 20 min (t2). The maximal value in each method was considered. SETTING: ICU, Hospital A.C. Camargo, Sao Paulo, Brazil. PATIENTS: Fifty-four consecutive patients undergoing short-term mechanical ventilation who became eligible for the study when their physicians decided to restore spontaneous breathing. RESULTS: MIPbest values were arrived at using MIPuni 75% of the time either in t1 or t2. MIPuni yielded a higher average of MIPbest values in t1 and t2 ($p < 0.0001$). The effort-to-effort coefficient of variation of one method compared with the other during t1 and t2 was similar ($p > 0.2$ for t1; $p > 0.8$ for t2). Also, when comparing t1 and t2, the coefficients of variation were similar for each method ($p > 0.62$). CONCLUSIONS: Because MIPuni displayed the maximal values, it is the best method for estimating MIP in patients undergoing short-term mechanical ventilation. The reproducibility of consecutive measurements was similar between the methods, even after a short period of time.

Evaluation of Electrical Impedance Tomography in the Measurement of PEEP-Induced Changes in Lung Volume—Kunst PW, de Vries PM, Postmus PE, Bakker J. Chest 1999 Apr;115(4):1102–1106.

STUDY OBJECTIVES: A new noninvasive practical technique called electrical impedance tomography (EIT) was examined for the measurement of alveolar recruitment. DESIGN: Prospective clinical study. SETTING: ICU of a general hospital. PATIENTS: Acute respiratory failure (ARF) patients. MEASUREMENTS: The ventilation-induced impedance changes (VICs) of the nondependent and the dependent part of the lung were determined by EIT as a measure of tidal volume distribution. By the use of an impedance ratio (IR), defined as the VIC of the nondependent part of the lung divided by the VIC of the dependent part of the lung, the ventilation performances in both parts of the lung were compared to each other. RESULTS: Between patients, the VIC of the nondependent part of the lung was significantly lower in the patients with a level of positive end-expiratory pressure (PEEP) of > 10 cm H_2O than in patients with a PEEP of < 5 cm H_2O ($p < 0.05$). A significantly lower IR (-/+ SD) was found in the group with PEEP of > 10 cm H_2O than in the group with PEEP between 0 and 5 cm H_2O (1.28 ± 0.58 vs 2.99 ± 1.24, respectively; $p < 0.01$). In individual patients, the VIC of the whole lung increased when the PEEP level was increased. The VICs of the nondependent part of the lung and of the dependent part of the lung showed significant increases at a PEEP of 10 cm H_2O compared to a PEEP of 0 cm H_2O ($p < 0.05$). Also the IR decreased in individual patients when the PEEP was increased; a significant decrease was found at 10 cm H_2O compared to 0 cm H_2O (1.67 ± 1.24 vs 2.23 ± 1.47, respectively; $p < 0.05$). CONCLUSIONS: The decrease in IR indicates an increase in VIC in the dependent part of the lung above the nondependent part of the lung. The increase in VIC can be regarded as an increase in lung volume, implying alveolar recruitment in the dependent part of the lung. The same results also have been shown in earlier reports by CT scan. Since EIT is far more practical than CT scanning and also is a bedside method, EIT might help in the adjustment of ventilator settings in ARF patients.

The Potential for Dissemination of Mycobacterium Tuberculosis Through the Anesthesia Breathing Circuit—Langevin PB, Rand KH, Layon AJ. Chest 1999 Apr;115(4):1107–1114.

BACKGROUND: Respiratory pathogens that pass through the anesthesia breathing system potentially can infect other patients. This study was designed to determine if bacteria can pass through contemporary anesthesia breathing systems and if the environment within the machine is hostile to these organisms. METHODS: *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Mycobacterium tuberculosis* were nebulized into the expiratory limb of an anesthesia breathing circuit and collected from the inspiratory and expiratory limbs in an impinger system that provided a quantitative determination of the number of organisms entering the circuit and the number that would reach the patient in the inspiratory gas. Bacteria were collected before, during, and after nebulization. A second experiment determined if a saturated solution of soda lime was bactericidal. RESULTS: When the gas flow through the circuit was interrupted for < 1 h following the nebulization period, large numbers of microorganisms ($1 \text{ x } 10^3$ to $1 \text{ x } 10^5$, around 100% of the nebulized organisms) were collected from the inspiratory gas. Soda lime itself was not bactericidal for any of the organisms tested, but solutions of this material with a pH of 12 were bactericidal. CONCLUSION: Cross contamination between patients may occur unless the gas flow through the anesthesia breathing system is interrupted for > 1 h.

The Upper Airway Resistance Syndrome (review)—Exar EN, Collop NA. Chest 1999 Apr; 115(4):1127–1139.

The upper airway resistance syndrome (UARS) is a recently described form of sleep-disordered breathing in which repetitive increases in resistance to airflow within the upper airway lead to brief arousals and daytime somnolence. This review will first describe the chronological progression of our understanding of UARS within the broader context of sleep-disordered breathing. The primary symptom, daytime somnolence, appears to result directly from repetitive EEG arousals. The level of negative intrathoracic pressure is the most likely stimulus for arousal, possibly mediated by mechanoreceptors in the upper airway. A general consensus regarding the exact clinical definitions and the physiologic measurement techniques leading to a diagnosis does not exist, although esophageal manometry and pneumotachographic airflow measurements taken during polysomnography are the "gold standard." Less invasive diagnostic modalities have been proposed, but none of them have been well-validated. Aside from daytime somnolence, hypertension is an important sequela of this disorder, likely resulting from autonomic and cardiovascular changes induced by increased negative intrathoracic pressure. Nasal continuous positive airway pressure is the most efficacious form of therapy, although low patient compliance may limit its practical application. The safety and efficacy of surgical treatments are poorly documented in the literature. Palatal tissue reduction by radiofrequency ablation and the use of oral appliances hold promise as safe and effective modalities, but these treatments require further study.

Effects of Writing About Stressful Experiences on Symptom Reduction in Patients with Asthma or Rheumatoid Arthritis: A Randomized Trial—Smyth JM, Stone AA, Hurewitz A, Kaell A. JAMA 1999 Apr 14;281(14): 1304–1309.

CONTEXT: Nonpharmacological treatments with little patient cost or risk are useful supplements to pharmacotherapy in the treatment of patients with chronic illness. Research has demonstrated that writing about emotionally traumatic experiences has a surprisingly beneficial effect on symptom reports, well-being, and health care use in healthy individuals. OBJECTIVE: To determine if writing about stressful life experiences affects disease status in patients with asthma or rheumatoid arthritis using standardized quantitative outcome measures. DESIGN: Randomized controlled trial conducted between October 1996 and December 1997. SETTING: Outpatient community residents drawn from private and institutional practice. PATIENTS: Volunteer sample of 112 patients with asthma ($n = 61$) or rheumatoid arthritis (n = 51) received the intervention; 107 completed the study, 58 in the asthma group and 49 in the rheumatoid arthritis group. INTERVENTION: Patients were assigned to write either about the most stressful event of their lives (n = 71; 39 asthma, 32 rheumatoid arthritis) or about emotionally neutral topics (n = 41; 22 asthma, 19 rheumatoid arthritis) (the control intervention). MAIN OUTCOME MEASURES: Asthma patients were evaluated with spirometry and rheumatoid arthritis patients were clinically examined by a rheumatologist. Assessments were conducted at baseline and at 2 weeks and 2 months and 4 months after writing and were done blind to experimental condition. RESULTS: Of evaluable patients 4 months after treatment, asthma patients in the experimental group showed improvements in lung function (the mean percentage of predicted forced expiratory volume in 1 second [FEV_1] improved from 63.9% at baseline to 76.3% at the 4-month follow-up; $p < 0.001$), whereas control group patients showed no change. Rheumatoid arthritis patients in the experimental group showed improvements in overall disease activity (a mean reduction in disease severity from 1.65 to 1.19 [28%] on a scale of 0 [asymptomatic] to 4 [very severe] at the 4-month follow-up; $p = 0.001$), whereas control group patients did not change. Combining all completing patients, 33 (47.1%) of 70 experimental patients had clinically relevant improvement, whereas 9 (24.3%) of 37 control patients had improvement ($P=.001$). CONCLUSION: Patients with mild to moderately severe asthma or rheumatoid arthritis who wrote about stressful life experiences had clinically relevant changes in health status at 4 months compared with those in the control group. These gains were beyond those attributable to the standard medical care that all participants were receiving. It remains unknown whether these health improvements will persist beyond 4 months or whether this exercise will prove effective with other diseases. *See the related editorial: Spiegel D.* ***Healing Words: Emotional Expression and Disease Outcome.*** *JAMA 1999 Apr 14;281(14):1328–1329.*

The Association Between Hospital Volume and Survival After Acute Myocardial Infarction in Elderly Patients—Thiemann DR, Coresh J, Oetgen WJ, Powe NR. N Engl J Med 1999 May 27;340(21):1640–1648.

BACKGROUND: Patients with chest pain thought to be due to acute coronary ischemia are typically taken by ambulance to the nearest hospital. The potential benefit of field triage directly to a hospital that treats a large number of patients with myocardial infarction is unknown. METHODS: We conducted a retrospective cohort study of the relation between the number of Medicare patients with myocardial infarction that each hospital in the study treated (hospital volume) and long-term survival among 98,898 Medicare patients 65 years of age or older. We used proportional-hazards methods to adjust for clinical, demographic, and health-system-related variables, including the availability of invasive procedures, the specialty of the attending physician, and the area of residence of the patient (rural, urban, or metropolitan). RESULTS: The patients in the quartile admitted to hospitals with the lowest volume were 17 percent more likely to die within 30 days after admission than patients in the quartile admitted to hospitals with the highest volume (hazard ratio, 1.17; 95 percent confidence interval, 1.09 to 1.26; $p<0.001$), which resulted in 2.3 more deaths per 100 patients. The crude mortality rate at one year was 29.8 percent among the patients admitted to the lowest-volume hospitals, as compared with 27.0 percent among those admitted to the highest-volume hospitals. There was a continuous inverse dose-response relation between hospital volume and the risk of death. In an analysis of subgroups defined according to age, history of cardiac disease, Killip class of infarction, presence or absence of contraindications to thrombolytic therapy, and time from the onset of symptoms, survival at high-volume hospitals was consistently better than at low-volume hospitals. The availability of technology for angioplasty and bypass surgery was not independently associated with overall mortality. CONCLUSIONS: Patients with acute myocardial infarction who are admitted directly to hospitals that have more experience treating myocardial infarction, as reflected by their case volume, are more likely to survive than are patients admitted to low-volume hospitals. *See the related editorial: Hannan EL.* ***The Relation Between Volume and Outcome in Health Care.*** *N Engl J Med 1999 May 27;340(21):1677–1679.*

INGMAR MEDICAL

Expand Your Vision with a Complete Family of Solutions For Lung Simulation

Hands-on Instruction

Advanced Research Applications

Neonatal to Adult

Passive and Active Lung Models

IngMar Medical ... Expanding the Vision of Respiratory Care

Tel: 800-583-9910 Fax: 412-683-8404
Internet: http://www.ingmarmed.com
Email: info@ingmarmed.com

Circle 124 on reader service card

Are Guidelines Following Guidelines? The Methodological Quality of Clinical Practice Guidelines in the Peer-Reviewed Medical Literature—Shaneyfelt TM, Mayo-Smith MF, Rothwangl J. JAMA 1999 May 26;281(20): 1900–1905.

CONTEXT: Practice guidelines play an important role in medicine. Methodological principles have been formulated to guide their development. OBJECTIVE: To determine whether practice guidelines in peer-reviewed medical literature adhered to established methodological standards for practice guidelines. DESIGN: Structured review of guidelines published from 1985 through June 1997 identified by a MEDLINE search. MAIN OUTCOME MEASURES: Mean number of standards met based on a 25-item instrument and frequency of adherence. RESULTS: We evaluated 279 guidelines, published from 1985 through June 1997, produced by 69 different developers. Mean overall adherence to standards by each guideline was 43.1% (10.77/25). Mean (SD) adherence to methodological standards on guideline development and format was 51.1% (25.3%); on identification and summary of evidence, 33.6% (29.9%); and on the formulation of recommendations, 46% (45%). Mean adherence to standards by each guideline improved from 36.9% (9.2/25) in 1985 to 50.4% (12.6/25) in 1997 ($p < 0.001$). However, there was little improvement over time in adherence to standards on identification and summary of evidence from 34.6% prior to 1990 to 36.1% after 1995 ($p = 0.11$). There was no difference in the mean number of standards satisfied by guidelines produced by subspecialty medical societies, general medical societies, or government agencies ($p = 0.55$). Guideline length was positively correlated with adherence to methodological standards ($p = 0.001$). CONCLUSION: Guidelines published in the peer-reviewed medical literature during the past decade do not adhere well to established methodological standards. While all areas of guideline development need improvement, greatest improvement is needed in the identification, evaluation, and synthesis of the scientific evidence.

Tourism and Hotel Revenues Before and After Passage of Smoke-Free Restaurant Ordinances—Glantz SA, Charlesworth A. JAMA 1999 May 26;281(20):1911–1918.

CONTEXT: Claims that ordinances requiring smoke-free restaurants will adversely affect tourism have been used to argue against passing such ordinances. Data exist regarding the validity of these claims. OBJECTIVE: To determine the changes in hotel revenues and international tourism after passage of smoke-free restaurant ordinances in locales where the effect has been debated. DESIGN: Comparison of hotel revenues and tourism rates before and after passage of 100% smoke-free restaurant ordinances and comparison with US hotel revenue overall. SETTING: Three states (California, Utah, and Vermont) and 6 cities (Boulder, Colo; Flagstaff, Ariz; Los Angeles, Calif; Mesa, Ariz; New York, NY; and San Francisco, Calif) in which the effect on tourism of smoke-free restaurant ordinances had been debated. MAIN OUTCOME MEASURES: Hotel room revenues and hotel revenues as a fraction of total retail sales compared with preordinance revenues and overall US revenues. RESULTS: In constant 1997 dollars, passage of the smoke-free restaurant ordinance was associated with a statistically significant increase in the rate of change of hotel revenues in 4 localities, no significant change in 4 localities, and a significant slowing in the rate of increase (but not a decrease) in 1 locality. There was no significant change in the rate of change of hotel revenues as a fraction of total retail sales ($p=0.16$) or total US hotel revenues associated with the ordinances when pooled across all localities ($p = 0.93$). International tourism was either unaffected or increased following implementation of the smoke-free ordinances. CONCLUSION: Smoke-free ordinances do not appear to adversely affect, and may increase, tourist business.

Neurohormonal Responses During Positive Pressure Mechanical Ventilation—Frazier SK. Heart Lung 1999 May-Jun;28(3):149–165.

Positive pressure mechanical ventilation is used daily in critical care units to support ventilation and improve oxygenation in critically ill patients. One adverse response to positive pressure mechanical ventilation is a reduction in urinary output and sodium and water retention. This consequence is attributed to complex neurohormonal responses intended to maintain hemodynamic homeostasis. This article reviews the physiologic nature of these responses and research findings related to these responses and provides clinicians with information about the importance of these responses, particularly in patients with underlying cardiac dysfunction.

An Evaluation of Facial Expression Displayed by Patients with Chest Pain—Dalton JA, Brown L, Carlson J, McNutt R, Greer SM. Heart Lung 1999 May-Jun;28(3):168–174.

OBJECTIVE: To determine whether patterns of facial expressions of patients complaining of chest pain can aid in the diagnosis of myocardial infarctions. DESIGN: Exploratory. SETTING: Southeastern university medical center emergency department (ED). PATIENTS: Twenty-eight patients in the ED who presented with chest pain. Age range was 40 to 84 years, with a mean of 65.4 years.OUTCOME MEASURES: Common patterns of facial expressions among patients diagnosed with myocardial infarction. INTERVENTION: On admission to the ED, patients were videotaped during physical examination. Videotapes were reviewed to code the facial action units exhibited by the patients. The presence or absence of creatine kinase (CK) enzymes was determined; these results were blinded to the coders until after all facial actions were coded. RESULTS: With use of the Facial Action Coding System, 4 facial expressions were found to be associated with true myocardial infarction: lowering the brow, pressing the lips, parting the lips, and turning the head left. CONCLUSION: Additional research with a larger sample of patients presenting with chest pain is needed to validate the findings of this study. However, if these findings are supported, instruction regarding assessment of specific facial expressions in clinical settings may be warranted.

Patients' and Family Members' Perceptions of Transfer from Intensive Care—Leith BA. Heart Lung 1999 May-Jun;28(3):210–218.

OBJECTIVE: To describe patients' and family members' perceptions of transfer from an intensive care unit (ICU). DESIGN: Qualitative component of a descriptive, cross-sectional survey. SETTING: Two university-affiliated tertiary care centres in western Canada. PARTICIPANTS: Fifty-three patients and 35 family members who had been transferred from a medical ICU within the previous 48 hours. MEASURES: Content analysis of responses to 3 open-ended questions relating to transfer from the ICU. RESULTS: Patients and family members had 3 major responses of transfer from the ICU: positive, neutral or ambivalent, and negative. Although some patients and family members perceived the transfer from the ICU as a sign of progress, many individuals expressed concern about the sudden and dramatic change in the level of care after transfer. CONCLUSION: Patients and family members perceived the transfer from the ICU as a significant and sometimes negative event.

Difficult Patient Encounters in the Ambulatory Clinic: Clinical Predictors and Outcomes—Jackson JL, Kroenke K. Arch Intern Med 1999 May 24;159(10):1069–1075.

BACKGROUND: One sixth of patient encounters are perceived as difficult by clinicians. Our goal was to assess clinical predictors and outcomes from such encounters. METHODS: Five hundred adults presenting to a primary care walk-in clinic with a physical symptom completed surveys before the visit, immediately after the visit, at 2 weeks, and at 3 months. Patient measurements included mental disorders (PRIME-MD), functional status (Medical Outcomes Study Short-Form Health Survey [SF-6]), satisfaction (RAND 9-item survey), symptom resolution, visit costs, previsit and residual expectations of care, and health services utilization. Measurements from the 38 participating clinicians included the Physician's Belief Scale and physician perception of encounter difficulty (Difficult Doctor-Patient Relationship Questionnaire). RESULTS: Seventy-four patient encounters (15%) were rated as difficult. Patients in such encounters were more likely to have a mental disorder (odds ratio, 2.4; 95% confidence interval, 1.3–4.4), more than 5 somatic symptoms (odds ratio, 1.4; 95% confidence interval, 1.1–1.8), and more severe symptoms (odds ratio, 1.6; 95% confidence interval, 1.04–2.3). Difficult-encounter patients had poorer functional status, more unmet expectations ($p = 0.005$), less satisfaction with care ($p = 0.03$), and higher use of health services ($p < 0.001$). Clinicians with poorer psychosocial attitudes as reflected by higher scores on the Physician's Belief Scale experienced more encounters as being difficult (23% vs 8%; p< 0.001). CONCLUSIONS: Patients presenting with physical symptoms who are perceived as difficult are more likely to have a depressive or anxiety disorder, poorer functional status, unmet expectations, reduced satisfaction, and greater use of health care services. Physicians with poorer psychosocial attitudes are more likely to experience encounters as difficult.

Diagnosing Pneumonia by Physical Examination: Relevant or Relic?—Wipf JE, Lipsky BA, Hirschmann JV, Boyko EJ, Takasugi J, Peugeot RL, Davis CL. Arch Intern Med 1999 May 24;159(10):1082–1087.

BACKGROUND: The reliability of chest physical examination and the degree of agreement among examiners in diagnosing pneumonia based on these findings are largely unknown. OBJECTIVES: To determine the accuracy of various physical examination maneuvers in diagnosing pneumonia and to compare the interobserver reliability of the maneuvers among 3 examiners. METHODS: Fifty-two male patients presenting to the emergency department of a university-affiliated Veterans Affairs medical center with symptoms of lower respiratory tract infection (cough and change in sputum) were prospectively examined. A comprehensive lung physical examination was performed sequentially by 3 physicians who were blind to clinical history, laboratory findings, and x-ray results. Examination findings by lung site and whether the examiner diagnosed pneumonia were recorded on a standard form. Chest x-ray films were read by a radiologist. RESULTS: Twenty-four patients had pneumonia confirmed by chest x-ray films. Twenty-eight patients did not have pneumonia. Abnormal lung sounds were common in both groups; the most frequently detected were rales in the upright seated position and bronchial breath sounds. Relatively high agreement among examiners (kappa approximately 0.5) occurred for rales in the lateral decubitus position and for wheezes. The 3 examiners' clinical diagnosis of pneumonia had a sensitivity of 47% to 69% and specificity of 58% to 75%. CONCLUSIONS: The degree of interobserver agreement was highly variable for different physical examination findings. The most valuable examination maneuvers in detecting pneumonia were unilateral rales and rales in the lateral decubitus position. The traditional chest physical examination is not sufficiently accurate on its own to confirm or exclude the diagnosis of pneumonia.

The Association Between Occupation and Asthma in General Medical Practice—Blanc PD, Eisner MD, Israel L, Yelin EH. Chest 1999 May;115(5):1259–1264.

BACKGROUND: In general practice settings, the proportion of adult asthma attributable to occupational factors is not known. OBJECTIVE: The goal of this study was to estimate the proportion of adult asthma cases that can be attributed to occupational factors initiating new disease onset and exacerbating preexisting disease. METHODS: We performed a cross-sectional analysis of interview data for 150 adults with asthma recruited from a random sample of family practice specialists. We ascertained the asthma and work histories of the subjects and estimated the proportion with likely work-initiated asthma and work-related asthma recrudescence. RESULTS: Seventy-four subjects (49%) reported adult-onset asthma while employed; an additional 25 (17%) reported recrudescence of previously quiescent childhood-onset asthma during employment. Of those with new-onset asthma while employed, 15 (10% of the study group; 95% confidence interval, 5 to 15%) were employed in occupations at increased risk of occupational asthma initiation on the basis of an independent job scoring matrix. Of those with asthma recrudescence in adulthood, seven (5% of the study group; 95% confidence interval, 2 to 8%) were employed in occupations at increased risk of exposures aggravating asthma. CONCLUSIONS: Among adults with asthma treated in general practice settings, > 1 in 10 patients has a work history strongly suggestive of a potential relationship between exposure and disease.

RESEARCH PNEUMOTACH SYSTEM
complete flow measurement system instrumentation module

RSS 100HR Research Pneumotach System with Hans Rudolph linear pneumotach

- **Model RSS 100HR** for the Hans Rudolph Linear & Bi-directional screen Pneumotachs in 8 flow ranges
- **Model RSS 100** For the Fixed-Orifice Pneumotachs available in Adult & Neonatal
- Measures gas flow using the **differential-pressure method**
- **Windows™ software** for displaying, storing and reviewing data
- System is fully functional as a **stand-alone device**
- **LCD screen** displays waveform and parameter data in real time
- **Embedded microprocessor** calculates flow rates and provides **correction for gas density, viscosity, temperature, barometric pressure,** and **airway pressure**
- Calculates over **15 ventilatory parameters**
- Integrated **breath detection** algorithm automatically detects start and end of breath
- Built-in **linearization routine calibration function** for Rudolph Pneumotachs
- **Serial RS232** and **analog outputs** provided for data collection

HANS RUDOLPH, inc.
MAKERS OF RESPIRATORY VALVES SINCE 1938
TEL: (816) 363-5522 U.S.A. & CANADA (800) 456-6695
FAX: 816-822-1414 E-Mail: hri@rudolphkc.com www.rudolphkc.com
7205 CENTRAL, KANSAS CITY, MISSOURI 64114 U.S.A.

Circle 120 on reader service card

Validation of a Standardized Version of the Asthma Quality of Life Questionnaire—Juniper EF, Buist AS, Cox FM, Ferrie PJ, King DR. Chest 1999 May;115(5):1265–1270.

BACKGROUND: In the original 32-item Asthma Quality of Life Questionnaire (AQLQ), five activity questions are selected by patients themselves. However, for long-term studies and large clinical trials, generic activities may be more appropriate. METHODS: For the standardized version of the AQLQ, the AQLQ(S), we formulated five generic activities (strenuous exercise, moderate exercise, work-related activities, social activities, and sleep) to replace the five patient-specific activities in the AQLQ. In a 9-week observational study, we compared the AQLQ with the AQLQ(S) and examined their measurement properties. Forty symptomatic adult asthma patients completed the AQLQ(S), the AQLQ, the Medical Outcomes Survey Short Form 36, the Asthma Control Questionnaire, and spirometry at baseline, 1, 5, and 9 weeks. RESULTS: Activity domain scores (mean ± SD) were lower with the AQLQ (5.7 ± 0.9) than with the AQLQ(S) (5.9 ± 0.8; p = 0.0003) and correlation between the two was moderate (r = 0.77). However, for overall scores, there was minimal difference (AQLQ, 5.4 ± 0.8; AQLQ(S), 5.5 ± 0.8; r = 0.99). Reliability (AQLQ intraclass correlation coefficient, 0.95; AQLQ(S) intraclass correlation coefficient, 0.96) and responsiveness (AQLQ, $p < 0.0001$; AQLQ(S), $p < 0.0001$) were similar for the two instruments. Construct validity (correlation with other measures of health status and clinical asthma) was also similar for the two instruments. CONCLUSIONS: The AQLQ(S) has strong measurement properties and is valid for measuring health-related quality of life in asthma. The choice of instrument should depend on the task at hand.

Reduction in Ventilator Response to CO_2 with Relaxation Feedback During CO_2 Rebreathing in Normal Adults—Holliday JE, Veremakis C. Chest 1999 May;115(5):1285–1292.

BACKGROUND: Previous studies have shown that relaxation biofeedback reduced time on the ventilator for the difficult-to-wean patients. OBJECTIVE: To test the hypothesis that the underlying mechanism of biofeedback ventilator weaning was the reduction of neural respiratory drive (NRD). DESIGN: Prospective, linear regression analysis. SETTING: Critical care medicine department in tertiary health care hospital. SUBJECTS: Fifteen healthy adult volunteers were randomly assigned to the biofeedback group, and 15 healthy adult volunteers were randomly assigned to a control group. INTERVENTIONS: Relaxation feedback was administered while a single variable, P_{aCO_2}, was inputted to the respiratory control system and the output measured. While rebreathing 7% CO_2/93% O_2, the biofeedback group received a baseline session and a relaxation feedback session and the control group received a baseline session and a no feedback session. MEASUREMENTS AND RESULTS: During relaxation feedback, there was a significant ($p < 0.001$ to $p < 0.05$) reduction in the slope of minute ventilation ($\dot{V}_I$), mean inspiratory flow (V_T/T_I), occlusion pressure in 0.1 s from onset of inspiration (P_{100}), respiration rate (RR), and diaphragm (DA) EMG compared to baseline. We also found the above breathing parameters decreased significantly for relaxation feedback ($p < 0.001$–0.05), compared to baseline, at maximum end-tidal CO_2 (64 ± 1.2 mm Hg) (all data are expressed as mean ± SE). The decrease for $\dot{V}_I = -4.65 \pm 1.17$ L/min, DA EMG $= -0.4 \pm 0.21$ microV, $P_{100} = -1.13 \pm 0.56$ cm H_2O, $V_T/T_I = -144 \pm 82.91$ mL/s, and RR $= -3.1 \pm 0.79$ breaths/min. No significant changes occurred in these parameters for the control group. CONCLUSIONS: We conclude that the addition of the behavioral input of relaxation feedback results in decreasing the values of respiratory parameters that reflect NRD.

Ventilatory Drive at Rest and Perception of Exertional Dyspnea in Severe COPD—Marin JM, Montes de Oca M, Rassulo J, Celli BR. Chest 1999 May;115(5):1293–1300.

BACKGROUND: The reasons for exertional dyspnea in severe COPD are not well established, but they are not solely related to the mechanical load. We tested the hypothesis that breathlessness may be determined, in part, by the response of an individual's central output. METHODS: In 26 patients with severe COPD ($FEV_1 < 50\%$ predicted) and 22 matched control subjects, we assessed at rest the ventilatory and mouth occlusion pressure ($P_{0.1}$) response to hyperoxic progressive hypercapnia. At rest and during a symptom-limited exercise test, routine cardiopulmonary variables were measured, and respiratory muscle function was evaluated using esophageal and gastric pressure. Dyspnea was assessed with a visual analog scale. RESULTS: Dyspnea with or without leg discomfort limited exercise in 73% of patients. Peak exercise dyspnea correlated only with dyspnea at rest ($r = 0.5$, $p < 0.008$) and $P_{0.1}$ response to CO_2 ($\Delta P_{0.1}/\Delta$[end-tidal PCO_2]$P_{ET}CO_2$) ($r = 0.48$, $p = 0.02$). Multiple regression analysis including resting and exercise data as independent variables revealed that 47% of the variance for dyspnea at peak exercise was explained by a model including dyspnea at rest and $\Delta P_{0.1}/\Delta P_{ET}CO_2$. Again, $\Delta P_{0.1}/\Delta P_{ET}CO_2$ was the only predictor for the change in dyspnea from rest to peak exercise (Δ Dyspnea, $r^2 = 0.28$, $p = 0.005$). There was no correlation between exercise dyspnea and any metabolic variable, pulmonary function, or respiratory muscle function test. CONCLUSION: In severe COPD, exertional dyspnea is not simply related to respiratory muscle load or mechanical impairment, but also to an individual's central motoneural output to the respiratory system.

Determination of Hemoglobin Saturation in Patients with Acute Sickle Chest Syndrome: A Comparison of Arterial Blood Gases and Pulse Oximetry—Kress JP, Pohlman AS, Hall JB. Chest 1999 May;115(5):1316–1320.

STUDY OBJECTIVES: To evaluate three different methods of measuring oxygen saturation in patients suffering from acute sickle chest syndrome. DESIGN: A prospective, descriptive study of 9 months' duration. SETTING: A tertiary care university hospital. PATIENTS: Adult patients with acute sickle chest syndrome scheduled to undergo RBC exchange transfusion. INTERVENTIONS: None. MEASUREMENTS: Baseline hemoglobin oxygen saturation was determined simultaneously by (1) calculation based on P_{aO_2} and an oxyhemoglobin dissociation curve algorithm, (2) co-oximetry, and (3) pulse oximetry. These same measures were repeated after exchange transfusion. Baseline and postexchange hemoglobin electrophoresis was performed in all patients. RESULTS: Baseline calculated saturation overestimated true saturation (determined by co-oximetry) with a baseline mean bias (co-oximetry minus calculated saturation) of -6.78 ± 2.63% (95% confidence interval for bias: −8.37% to −5.19%). Pulse oximetry was not different than co-oximetry at baseline with a baseline bias of +1.86 ± 3.25% (95% confidence interval: −0.1% to 3.82%). After exchange transfusion, there was no bias between either co-oximetry and calculated saturation (mean difference: −0.17 ± 1.31% [95% confidence interval: −0.95% to 0.61%]), or co-oximetry and pulse oximetry (mean difference: +0.3 ± 1.53% [95% confidence interval: −0.62% to 1.22%]). CONCLUSIONS: Calculated saturation overestimates true saturation during acute sickle chest syndrome. This discrepancy abates after exchange transfusion. Pulse oximetry more closely follows co-oximetry than does calculated saturation during acute sickle chest syndrome.

Effect of Ipratropium Bromide Treatment on Oxygen Saturation and Sleep Quality in COPD—Martin RJ, Bartelson BL, Smith P, Hudgel DW, Lewis D, Pohl G, et al. Chest 1999 May;115(5):1338–1345.

STUDY OBJECTIVES: Patients with COPD are at risk of experiencing a deterioration in arterial oxygen saturation (S_{aO_2}) during sleep, which is generally most pronounced during rapid eye movement (REM) sleep. Increased cholinergic tone has been suggested as a contributing factor to this decrease in S_{aO_2}. Therefore, we investigated whether 4-week treatment with ipratropium bromide inhalation solution 0.02% (qid) could improve sleep characteristics in COPD. DESIGN: Randomized, placebo-controlled, double-blind, two-arm parallel study of 4 weeks of treatment with ipratropium bromide solution or placebo. SETTING: Multicenter investigation. PATIENTS: Thirty-six patients with moderate-to-severe COPD ($FEV_1 < 65\%$ of predicted). MEASUREMENTS AND RESULTS: Evaluation included polysomnographic, pulmonary function, and subjective quality of sleep (visual analog scale [VAS]) assessments. It was found that 4 weeks of treatment with ipratropium bromide solution in patients with COPD led to the following: (1) a significant ($p = 0.05$) improvement in mean nocturnal S_{aO_2} with the more severe the nocturnal desaturation, the greater the improvement in S_{aO_2}; (2) significant ($p = 0.03$) improvement in perceived sleep quality (VAS: 5.5 ± 0.5 after placebo; 7.2 ± 0.5 after ipratropium); (3) a significant ($p = 0.05$) increase in REM sleep time (48.6 ± 6.3 min after placebo; 66.5 ± 6.4 min after ipratropium) with no effect on other sleep stages or total sleep time; and (4) a significant ($p = 0.01$) increase in pre-sleep FVC and flow rate at 50% of the vital capacity. CONCLUSIONS: These findings demonstrate that ipratropium bromide therapy can improve sleep S_{aO_2} as well as sleep quality in patients with moderate-to-severe COPD.

Handi™
Oxygen Analyzer
* Simple
* Affordable
* Reliable
* Pocket size

MSA Model*
Respiratory Sensor
MiniOX I, II, III
MiniOX 3000

MAXBlend
Air/Oxygen Mixer
* Unmatched performance and reliability.
* Simple operation.
* Low cost.

Siemens*
Respiratory Sensor
Models 900C & 300
Ventilators

maxtec,™ Inc. For more product information call: 800-748-5355
FORMERLY CERAMATEC

2425 South 900 West, Suite B, S.L.C., Utah 84119 phone: 800-748-5355 fax: 801-956-1002 www.maxtecinc.com
[illegible] MSA are registered trademarks of their respective corporations, maxtec is [illegible]

Circle 117 on reader service card

Tracheobronchial Foreign Bodies: Presentation and Management in Children and Adults—Baharloo F, Veyckemans F, Francis C, Biettlot MP, Rodenstein DO. Chest 1999 May;115(5):1357–1362.

STUDY OBJECTIVES: To compare the clinical and management aspects of tracheobronchial aspirated foreign body (AFB) removal in children and adults; to assess the influence of the operator's experience on the outcome of the procedure. DESIGN: A retrospective review of a 20-year experience (from 1976 to 1996). SETTING: A 900-bed university hospital. PATIENTS: Eighty-four children up to 8 years old (the child group) and 28 adult patients (the adult group). RESULTS: The peak incidence of foreign body aspiration occurred during the second year of life in the child group and during the sixth decade in the adult group. The symptoms at presentation were similar in both age groups, but the diagnosis was significantly delayed in the adults. The AFBs were lodged preferentially in the right bronchial tree only in the adults; a central location was predominant (but not at all exclusive) in the children. Atelectasis was more common in the adults, and air trapping was more common in the children. The most frequent procedure was rigid bronchoscopy; when a flexible bronchoscope was used, it was always in the adult patients. When the operator was less experienced, a failed first attempt at bronchoscopy and the need for a second procedure were significantly more frequent. CONCLUSIONS: At presentation, the symptoms seen with AFBs do not differ according to the age of the patient; however, the delay to diagnosis, the location of the AFBs, and the radiographic images differ between child and adult populations. The removal of AFBs in patients of all ages can be performed by the same operators. Because the outcome associated with these procedures improves when the operator is experienced, the removal of AFBs should be performed in medical centers that are capable of acquiring and maintaining the necessary expertise.

Risk Factors for an Outbreak of Multi-Drug-Resistant Acinetobacter Nosocomial Pneumonia Among Intubated Patients—Husni RN, Goldstein LS, Arroliga AC, Hall GS, Fatica C, Stoller JK, Gordon SM. Chest 1999 May;115(5):1378–1382.

INTRODUCTION: *Acinetobacter baumanii* is a Gram-negative coccobacillus that is normally a commensal pathogen but can be a nosocomial pathogen. An epidemiologic study was performed to investigate an outbreak of A baumanii that occurred in our medical intensive care unit (MICU) from March to September 1995. METHODS: A case-control study was performed by retrospective chart review, comparing case patients to randomly selected patients who were mechanically ventilated in the MICU for at least 1 week during the outbreak. A case patient was defined as any patient with an Acinetobacter infection in which the epidemic strain was considered to be a pathogen. The epidemic strain was defined by its antibiogram. Case patients and control patients were compared for age, gender, underlying disease, acute physiology and chronic health evaluation III score, length of MICU stay, prior antibiotic use, presence of fever, sepsis, type of pulmonary infiltrate, and outcome. Environmental and handwashing studies also were performed during the period of the outbreak. Molecular typing was performed on available bloodstream isolates. RESULTS: There were 15 cases of *A baumanii* nosocomial pneumonia. Fifty percent were bacteremic; one chart was unavailable for review. Twenty-nine patients were identified as control patients. The mean age for case patients was 50 (range, 21 to 84). The mean duration of time from admission to the ICU to infection was 12.8 days (range, 4 to 40). Sepsis developed in 35% of the case patients. Forty-three percent of the case patients died during their hospitalization, with two of those deaths attributed to Acinetobacter infection. Univariate analysis showed that prior use of ceftazidime was associated with infection with Acinetobacter (11/14 case patients compared to 11/29 control patients; $p < 0.01$). Pulsed-field gel electrophoresis revealed two strains to be responsible for the outbreak. Hand washing was performed before patient contact by only 10% of health-care workers, and only 32% washed their hands after patient contact. CONCLUSION: The use of ceftazidime was associated with an increased risk of nosocomial pneumonia with resistant strains of Acinetobacter. Health-care workers need to improve compliance with hand-washing recommendations.

Posterior Tracheal Wall Perforation During Percutaneous Dilational Tracheostomy: An Investigation into Its Mechanism and Prevention—Trottier SJ, Hazard PB, Sakabu SA, Levine JH, Troop BR, Thompson JA, McNary R. Chest 1999 May;115(5):1383–1389.

OBJECTIVES: Part 1: To describe the complication of posterior tracheal wall injury and perforation associated with the percutaneous dilational tracheostomy (PDT). Part 2: To determine the mechanism of posterior tracheal wall injury during PDT. DESIGN: Prospective observational study. SUBJECTS: Part 1: Medical-surgical ICU patients requiring tracheostomy. Part 2: Swine and cadaver models. INTERVENTIONS: Part 1: Consecutive medical-surgical ICU patients undergoing tracheostomy tube insertion via the percutaneous dilation technique with bronchoscopic guidance were enrolled in the study. Demographic data and complications were recorded. Part 2: Tracheostomy tubes were inserted via the percutaneous dilational technique in the swine model with concomitant bronchoscopic video recording from the proximal and distal airways. Tracheostomy tubes were inserted via the percutaneous dilational technique in the cadaver model followed by anatomic inspection of the airway. RESULTS: Part 1: Seven (29%) of 24 medical-surgical ICU patients sustained complications associated with PDT. Three patients (12.5%) sustained posterior tracheal wall perforations followed by the development of tension pneumothoraces. Part 2: The swine model demonstrated that posterior tracheal wall perforation may occur during PDT when the guiding catheter is withdrawn into the dilating catheters. Five-centimeter posterior tracheal wall mucosal lacerations occurred when the guidewire and the guiding catheter were not properly stabilized during PDT. CONCLUSION: Percutaneous dilational tracheostomy was associated with a 29% complication rate in this observational study. Of concern was the high rate (12.5%) of posterior tracheal wall perforation. The swine and cadaver models suggest that posterior tracheal wall injury or perforation may occur if the guidewire and guiding catheter are not properly stabilized. To avoid posterior tracheal wall injury, the guidewire and guiding catheter should be firmly stabilized during PDT.

Realizing the Promise: Delivering Pulmonary Continuing Medical Education over the Internet—Peterson MW, Galvin JR, Dayton C, D'Alessandro MP. Chest 1999 May;115(5):1429–1436.

STUDY OBJECTIVES: Continuing medical education (CME) is meant to bridge the gap between new scientific observations and clinical practice. However, traditional CME has not been effective at altering the behaviors of physicians. One reason for this failure of traditional CME programs may be their inflexibility. In traditional CME, the clinician does not choose the topic, the pace of the program, or the place of learning, and the CME material cannot be easily delivered to the point of care where the clinician needs the information. Computers and computer networks have the potential to accomplish these goals. CME has begun to appear on the Internet; however, there have been few evaluations of its usefulness, acceptance, and effectiveness. Over the last 18 months, we have developed three on-line pulmonary CME programs, and we have delivered them on the Virtual Hospital, the University of Iowa's digital health sciences library on the Internet. We report our initial experience with this CME material. DESIGN: We measured the frequency with which the Internet-delivered CME is accessed by monitoring page accessions and by using a log file analysis program (Analog 1.2.3; University of Cambridge Statistical Laboratory; Cambridge, UK). In addition, we collected all completed CME examinations and evaluation forms submitted by registered users. MEASUREMENTS AND RESULTS: We have found that the frequency with which the Internet-delivered CME is accessed has continued to increase with time (2.3-fold increase over 18 months), that evaluations of technical and content issues are strongly favorable, and that some clinicians have been willing to pay to receive CME through the medium of the Internet. CONCLUSIONS: We feel that with adequate peer review and quality control, physicians will use the Internet-delivered CME. However, several obstacles to wide use remain. These obstacles include issues regarding training in using the Internet for physicians, reluctance of physicians to participate in on-line commerce, and the current unavailability of CME to be delivered in small-grained quantities to the point of care. As these issues are addressed, we feel that on-line CME will represent an increasingly important CME medium for clinicians.

Patient Satisfaction with Conscious Sedation for Bronchoscopy—Putinati S, Ballerin L, Corbetta L, Trevisani L, Potena A. Chest 1999 May;115(5):1437–1440.

STUDY OBJECTIVE: Bronchoscopic technique is not standardized. Controversies exist with regard to premedication with sedatives before the test. To evaluate safety and efficacy of conscious sedation, we studied 100 randomized patients undergoing diagnostic bronchoscopy; patients received premedication with lidocaine spray and atropine sulfate i.m. (nonsedation group; 50 patients) or lidocaine spray, atropine i.m. and diazepam i.v. (sedation group; 50 patients). METHODS AND RESULTS: Monitoring during flexible fiberoptic bronchoscopy included continuous ECG and pulse oximetry. The procedure could not be completed in six patients. None received premedication with diazepam; among the patients who ended the examination, tolerance to the examination (visual analogue scale, 0 to 100; 0 = excellent; 100 = unbearable) was better in the sedation group. Low anxiety, male sex, but not age were also associated with improved patient tolerance to the test. Oxygen desaturation occurred in 17% of patients, and it was not more frequent after diazepam treatment. CONCLUSIONS: In our study, sedation had a beneficial effect on patient tolerance and rarely induced significant alterations in cardiorespiratory monitoring parameters.

Red Cell Distribution and the Recruitment of Pulmonary Diffusing Capacity—Hsia CC, Johnson RL Jr, Shah D. J Appl Physiol 1999 May;86(5):1460–1467.

The distribution of red blood cells in alveolar capillaries is typically nonuniform, as shown by intravital microscopy and in alveolar tissue fixed in situ. To determine the effects of red cell distribution on pulmonary diffusive gas transport, we computed the uptake of CO across a two-dimensional geometric capillary model containing a variable number of red blood cells. Red blood cells are spaced uniformly, randomly, or clustered without overlap within the capillary. Total CO diffusing capacity (D_{LCO}) and membrane diffusing capacity (D_{mCO}) are calculated by a finite-element method. Results show that distribution of red blood cells at a fixed hematocrit greatly affects capillary CO uptake. At any given average capillary red cell density, the uniform distribution of red blood cells yields the highest D_{mCO} and D_{LCO}, whereas the clustered distribution yields the lowest values. Random nonuniform distribution of red blood cells within a single capillary segment reduces diffusive CO uptake by up to 30%. Nonuniform distribution of red blood cells among separate capillary segments can reduce diffusive CO uptake by >50%. This analysis demonstrates that pulmonary microvascular recruitment for gas exchange does not depend solely on the number of patent capillaries or the hematocrit; simple redistribution of red blood cells within capillaries can potentially account for 50% of the observed physiological recruitment of D_{LCO} from rest to exercise.

GALILEO®

The new view of Ventilation

Derived from years of advanced research and practical clinical experience, HAMILTON MEDICAL introduces **GALILEO**, the most advanced ventilator in the world. **GALILEO** combines the newest innovative technology in an easy to operate system. Advanced Rules Based Strategic Modes combined (**Adaptive Support Ventilation ASV**) with Virtual Controls provides the maximum in patient care flexibility. **GALILEO** defines a new dimension in the ventilator management of critically ill patients today and into the future.

GALILEO is the ventilator of choice to implement comprehensive clinical pathways for all ventilator patients. Offering the clinician a new dimension in respiratory care, Ventilation without Limitations.

GALILEO is available from your local HAMILTON MEDICAL partner. For further information, contact HAMILTON MEDICAL at www.hamilton-medical.ch, phone +41-81-641-2627, fax +41-81-641-2689 or call our local representative.

Circle 119 on reader service card

HAMILTON MEDICAL
MAKING TECHNOLOGY SERVE MANKIND

International: HAMILTON MEDICAL AG, Via Nova, CH-7403 Rhäzüns/Switzerland, Telephone (+41) 81- 641 26 27, Fax (+41) 81- 641 26 89
USA: HAMILTON MEDICAL Inc., P.O.Box 30008, Reno, NV 89520, Telephone (702) 858-3201, (800) HAM-MED-1, Fax (702) 856-5621
Great Britain: HAMILTON MEDICAL (G.B.) Limited, Kimpton Business Centre, Kimpton Road, Sutton, Surrey SM3 9QR, Telephone 0181-641 9008, Fax 0181-641 9054
Germany: HAMILTON Deutschland GmbH, Abt. MEDICAL Daimlerweg 5a, D-64293 Darmstadt, Tel. (06151) 98020, Fax (06151) 89 17 33

Atopy in Children of Families with an Anthroposophic Lifestyle—Alm JS, Swartz J, Lilja G, Scheynius A, Pershagen G. Lancet 1999 May 1;353(9163):1485–1488.

BACKGROUND: Increased prevalence of atopic disorders in children may be associated with changes in types of childhood infections, vaccination programmes, and intestinal microflora. People who follow an anthroposophic way of life use antibiotics restrictively, have few vaccinations, and their diet usually contains live lactobacilli, which may affect the intestinal microflora. We aimed to study the prevalence of atopy in children from anthroposophic families and the influence of an anthroposophic lifestyle on atopy prevalence. METHODS: In a cross-sectional study, 295 children aged 5–13 years at two anthroposophic (Steiner) schools near Stockholm, Sweden, were compared with 380 children of the same age at two neighbouring schools in terms of history of atopic and infectious diseases, use of antibiotics and vaccinations, and social and environmental variables. Skin-prick tests were done for 13 common allergens, and we took blood samples from children and their parents for analysis of allergen-specific serum IgE-antibodies. FINDINGS: At the Steiner schools, 52% of the children had had antibiotics in the past, compared with 90% in the control schools. 18% and 93% of children, respectively, had had combined immunisation against measles, mumps, and rubella, and 61% of the children at the Steiner schools had had measles. Fermented vegetables, containing live lactobacilli, were consumed by 63% of the children at Steiner schools, compared with 4.5% at the control schools. Skin-prick tests and blood tests showed that the children from Steiner schools had lower prevalence of atopy than controls (odds ratio 0.62 [95% CI 0.43–0.91]). There was an inverse relation between the number of characteristic features of an anthroposophic lifestyle and risk of atopy (p for trend=0.01). INTERPRETATION: Prevalence of atopy is lower in children from anthroposophic families than in children from other families. Lifestyle factors associated with anthroposophy may lessen the risk of atopy in childhood. *See the related editorial:* ***Lifestyle and Atopy.*** *Strachan DP. Lancet 1999 May 1;353(9163):1457–1458.*

Systemic Adverse Effects of Inhaled Corticosteroid Therapy: A Systematic Review and Meta-Analysis—Lipworth BJ. Arch Intern Med 1999 May 10;159(9):941–955.

OBJECTIVE: To appraise the data on systemic adverse effects of inhaled corticosteroids. METHODS: A computerized database search from January 1, 1966, through July 31, 1998, using MEDLINE, EMBASE, and BIDS and using appropriate indexed terms. Reports dealing with the systemic effects of inhaled corticosteroids on adrenal gland, growth, bone, skin, and eye, and reports on pharmacology and pharmacokinetics were reviewed where appropriate. Studies were included that contained evaluable data on systemic effects in healthy volunteers as well as in asthmatic children and adults. A statistical meta-analysis using regression was performed for parameters of adrenal suppression in 27 studies. RESULTS: Marked adrenal suppression occurs with high doses of inhaled corticosteroid above 1.5 mg/d (0.75 mg/d for fluticasone propionate), although there is a considerable degree of interindividual susceptibility. Meta-analysis showed significantly greater potency for dose-related adrenal suppression with fluticasone compared with beclomethasone dipropionate, budesonide, or triamcinolone acetonide, whereas prednisolone and fluticasone propionate were approximately equivalent on a 10:1-mg basis. Inhaled corticosteroids in doses above 1.5 mg/d (0.75 mg/d for fluticasone propionate) may be associated with a significant reduction in bone density, although the risk for osteoporosis may be obviated by post-menopausal estrogen replacement therapy. Although medium-term growth studies showed suppressive effects with 400-microg/d beclomethasone dipropionate, there was no evidence to support any significant effects on final adult height. Long-term, high-dose inhaled corticosteroid exposure increases the risk for posterior subcapsular cataracts, and, to a much lesser degree, the risk for ocular hypertension and glaucoma. Skin bruising is most likely to occur with high-dose exposure, which correlates with the degree of adrenal suppression. CONCLUSIONS: All inhaled corticosteroids exhibit dose-related systemic adverse effects, although these are less than with a comparable dose of oral corticosteroids. Metaanalysis shows that fluticasone propionate exhibits greater dose-related systemic bioactivity compared with other available inhaled corticosteroids, particularly at doses above 0.8 mg/d. The long-term systemic burden will be minimized by always trying to achieve the lowest possible maintenance dose that is associated with optimal asthmatic control and quality of life.

The Role of the Primary Care Physician in Recognizing Obstructive Sleep Apnea—Kramer NR, Cook TE, Carlisle CC, Corwin RW, Millman RP. Arch Intern Med 1999 May 10;159(9):965–968.

BACKGROUND: Obstructive sleep apnea (OSA) is a common disorder among middle-aged adults. However, OSA is a recently described disorder for which most primary care physicians do not have formal training. The primary objectives of this article are to evaluate what percentage of patients referred by primary care physicians for sleep studies had OSA; to characterize the clinical features of these patients and compare them with our known OSA population; and to determine whether primary care physicians asked key questions contained in a work sheet to make the diagnosis of OSA. METHODS: A retrospective chart review at a hospital-based sleep center that is accredited to evaluate all sleep disorders, not just OSA. The health maintenance organization is a staff model one. PATIENTS: Sixty-nine patients who were referred for a sleep study by a health maintenance organization internist or family practitioner between June 1, 1994, and May 30, 1995. RESULTS: Ninety-six percent of the 68 patients referred for polysomnography had OSA. Most were very symptomatic and obese. These 68 patients represent 0.13% of the primary care patient panel. In addition, most of the patients were referred by a few physicians; 6 (11%) of the 55 physicians ordered 33% of the 68 studies. CONCLUSIONS: Primary care physicians did recognize obese patients with prominent symptoms of sleep apnea. However, only a small percentage of their patient panel was referred, suggesting that this condition is still underdiagnosed. This seems particularly true as most of the sleep studies were ordered by a small group of physicians. Future work incorporating educational interventions is necessary to improve detection and treatment of OSA.

Life-Threatening Events After Theophylline Overdose: A 10-Year Prospective Analysis—Shannon M. Arch Intern Med 1999 May 10; 159(9):989–994.

BACKGROUND: Despite the declining use of theophylline, episodes of intoxication continue to occur, producing seizures, arrhythmias, and death. OBJECTIVES: To further characterize major toxic effects and to examine the efficacy of existing interventions. METHODS: We conducted a longitudinal cohort study of patients with theophylline overdose. For a 125-month period, all patients referred to the Massachusetts Poison Control System in Boston with a serum theophylline concentration of 167 micromol/L (≥ 30 microg/mL) or more were followed up prospectively. Recommended management by the poison center was uniform and protocol based. RESULTS: Three hundred fifty-six patients were enrolled. Mean age was 34.5 years (range, 3 days to 98 years). Mean peak serum theophylline concentration was 336 micromol/L (60 microg/mL) (range, 167-1360 micromol/L [30–245 microg/mL]). One hundred sixty-two patients (45.5%) had acute, 144 (40.4%) had chronic, and 50 (14.0%) had acute-on-therapeutic poisoning. Seventy-four patients (20.8%) developed cardiac arrhythmias, and 29 (8.2%) developed seizures. Fifteen patients (4.2%) died, 11 (73%) of whom had chronic overmedication. Arrhythmias were significantly more common after chronic overmedication than after acute intoxication (35% vs. 10%; odds ratio, 4.97; 95% confidence interval, 2.68–9.23; $p < 0.001$). Eight percent of patients with

INFORMATION TO HELP YOUR PATIENTS BREATHE EASIER

Health Tips Fact Sheets

These one-page fact sheets help you share your knowledge and expertise on important issues in respiratory health with the community. Pad of 50.
$7.50 ($10.00 nonmembers)

Living with Asthma .... **Item R32**

Quiz Sheets

Inpatients and outpatients alike will find these "IQ Tests" challenging and informative. These true-and-false tests feature detailed answers on a variety of subjects vital to respiratory care. Pad of 50.
$7.50 ($10.00 nonmembers)

Check Your Asthma "IQ" .... **Item R60**

Asthma Disease State Management: Establishing a Partnership

A three-part video program that provides instruction in how to create an effective asthma disease management program in your facility and covers diagnosis, pharmacological therapy, environmental controls, patient/family education, and case studies. Approved for two hours of continuing education credit by CRCE and nursing CE credit. Includes workbook. 110 min.
Item PA101 $79.95 ($94.95 nonmembers)

INTERACTIVE CLINICAL SKILLS SOFTWARE

Status Asthmaticus Simulation

Involves the initial assessment of a 35-year-old man with a history of allergic asthma. Low-flow oxygen is administered initially, followed by bronchodilator therapy. As the simulation progresses, the patient is intubated and receives mechanical ventilation. The user is required to make initial settings and adjustments according to ABG results and patient response. Also included in the simulation is a switch to IMV mode, sedation, and eventual extubation and placement on a 40 percent aerosol mask.
Item SP12 $65

IISPs

Bronchodilators I: Sympathomimetic Amines

Understand the results of stimulating the autonomic nervous system and the use of sympathomimetic drugs to accomplish bronchodilation. Exposes you to basic aspects of adrenergic bronchodilators and the patient situations for which they are indicated.
Item CS14 $12 (nonmembers $16)

Bronchodilators II: Anti-Cholinergics and Xanthines

Identifies the three categories of drugs that promote bronchodilation and the mechanism of action for each. This package also provides examples of drugs in each category. Clinical situations are presented with methods of bronchodilation and rationale for method selected.
Item CS15 $12 (nonmembers $16)

Peak Flow Monitoring

Describes the method for performance of peak flow along with indications for peak flow monitoring in asthma. Discusses the minimal ATS recommendations for monitoring devices. Lists limitations of peak flow monitoring.
Item PE3 $12 (nonmembers $16)

VIDEOTAPES

Asthma Drugs and Medications: What's Right and What's Wrong

Reviews the pharmacology section of the revised NAEPP Guidelines. Additionally, the potential hazards and undesirable side effects of commonly prescribed medications will be discussed, with alternative treatment regimens suggested. Featuring William Luskin, MD, and David J. Pierson, MD.
Item VC81 $49.95 (nonmembers $99)

Asthma Disease Management: Using the Revised NAEPP Guidelines in Practice

Learn the four essential components of the NAEPP Guidelines that are essential to asthma disease management, and how these components are incorporated into a comprehensive asthma management program in the work setting, from the hospital to home care. Featuring Thomas J. Kallstrom, RRT, Gretchen Lawrence, BA, RRT, and Sam P. Giordano, MBA, RRT.
Item VC74 $49.95 (nonmembers $99)

To place your order, please call:

972-243-2272

Texas customers only, please add 8.25% sales tax (charged on product cost and shipping charges). Texas customers who are exempt from sales tax must supply an exemption certificate. MasterCard, Visa, Institutional Purchase Order or check payable to:

American Association for Respiratory Care
ATTN: Order Fulfillment Department
11030 Ables Lane • Dallas, Texas 75229
Phone 972-243-2272 Fax: 972-484-2720
www.aarc.org

Shipping Chart

$15 or Less	$4.25
$15.01 - $25.00	$5.25
$25.01 - $60.00	[illegible]
$60.01 - $80.00	$8.25
$80.01 - $150.00	$10.25
$150.01 - $250.00	$14.25
$251 or more	$16.25
International Orders Add $25	

chronic overmedication died compared with 2.5% of those with acute intoxication (odds ratio, 3.20; 95% confidence interval, 1.01–10.39; p = 0.04). There was no significant difference in the rate of major toxic effects (25.9% vs. 30.0%) or death (4.6% vs. 3.7%) among patients referred from 1986 to 1991 and from 1992 to 1996. CONCLUSIONS: Theophylline intoxication results in substantial morbidity and mortality, particularly in those with chronic overmedication. Treatment strategies fail to improve clinical outcome. With safer alternative options available, the current indications for theophylline should be rigorously evaluated with a goal toward minimal use of this agent.

Rapid Saline Infusion Produces Hyperchloremic Acidosis in Patients Undergoing Gynecologic Surgery—Scheingraber S, Rehm M, Sehmisch C, Finsterer U. Anesthesiology 1999 May;90(5):1265–1270.

BACKGROUND: Changes in acid-base balance caused by infusion of a 0.9% saline solution during anesthesia and surgery are poorly characterized. Therefore, the authors evaluated these phenomena in a dose-response study. METHODS: Two groups of 12 patients each who were undergoing major intraabdominal gynecologic surgery were assigned randomly to receive 0.9% saline or lactated Ringer's solution in a dosage of 30 $\text{mL} \times \text{kg}^{-1} \times \text{h}^{-1}$. The pH, arterial carbon dioxide tension, and serum concentrations of sodium, potassium, chloride, lactate, and total protein were measured in 30-min intervals. The serum bicarbonate concentration was calculated using the Henderson-Hasselbalch equation and also using the Stewart approach from the strong ion difference and the amount of weak plasma acid. The strong ion difference was calculated as serum sodium + serum potassium - serum chloride - serum lactate. The amount of weak plasma acid was calculated as the serum total protein concentration in g/dl x 2.43. RESULTS: Infusion of 0.9% saline, but not lactated Ringer's solution, caused a metabolic acidosis with hyperchloremia and a concomitant decrease in the strong ion difference. Calculating the serum bicarbonate concentration using the Henderson-Hasselbalch equation or the Stewart approach produced equivalent results. CONCLUSIONS: Infusion of approximately 30 $\text{mL} \times \text{kg}^{-1} \times \text{h}^{-1}$ saline during anesthesia and surgery inevitably leads to metabolic acidosis, which is not observed after administration of lactated Ringer's solution. The acidosis is associated with hyperchloremia. *See the related editorial: **Hyperchloremic Metabolic Acidosis Is a Predictable Consequence of Intraoperative Infusion of 0.9% Saline.** Prough DS, Bidani A. Anesthesiology 1999 May;90(5):1247–1249.*

Use of the Cuffed Oropharyngeal Airway As an Alternative to the Laryngeal Mask Airway with Positive-Pressure Ventilation—van Vlymen JM, Fu W, White PF, Klein KW, Griffin JD. Anesthesiology 1999 May;90(5):1306–1310.

BACKGROUND: The cuffed oropharyngeal airway is a modified Guedel-type oral airway with a cuff at its distal end. The objectives of this study were to compare the ability of the cuffed oropharyngeal airway and the laryngeal mask airway to provide positive-pressure ventilation during general anesthesia, and to assess their relative ease of use and ability to reduce total fresh gas flow rates. METHODS: In this prospective, randomized study, a cuffed oropharyngeal airway (n = 25) or a laryngeal mask airway (n = 25) device was inserted after induction of anesthesia intravenously using 2 mg/kg propofol. While anesthesia was maintained with sevoflurane and nitrous oxide, the leak pressure, leak fraction (the fractional difference between the inspired and expired tidal volume), minimum fresh gas flow rate, and need for airway manipulations were determined. The anesthesia provider who inserted the device completed an evaluation form at the end of the 15-min study period. RESULTS: Positive-pressure ventilation was established successfully on the first attempt in 92% of the patients when the cuffed oropharyngeal airway was used and in 88% of the patients when the laryngeal mask airway device was used. However, manipulations of the airway device were necessary more frequently (8 vs. 1 patient; $p < 0.05$) and the leak pressure was less (22 ± 6 cm water vs. 26 ± 5 cm water; $p < 0.05$) with the cuffed oropharyngeal airway than with the laryngeal mask airway. In addition, the leak fraction (0.19 ± 0.18 vs. 0.31 ± 0.22; $p < 0.05$) and the minimum fresh gas flow rate (1.3 ± 1.5 vs. 2.4 ± 2.5; p = 0.12) were less in the laryngeal mask airway group. CONCLUSIONS: Positive-pressure ventilation is possible with the laryngeal mask airway and cuffed oropharyngeal airway devices. Although the cuffed oropharyngeal airway can be inserted easily by inexperienced users with a high first-attempt success rate (> 90%), manipulations of the device may be required to maintain a patent airway. The laryngeal mask airway device allows positive-pressure ventilation at slightly greater peak inspiratory pressures.

Topical Lidocaine-Prilocaine Cream (EMLA) for Thoracostomy Tube Removal—Valenzuela RC, Rosen DA. Anesth Analg 1999 May;88(5):1107–1108.

We prospectively studied topical lidocaine-prilocaine cream (EMLA) versus IV morphine in a double-blinded, randomized fashion for pain relief during thoracostomy tube (chest tube; CT) removal. Adult patients who had undergone thoracotomy or median sternotomy were randomized to receive either EMLA cream over CT sites transdermally for 3 h or IV morphine 0.5 h before CT removal. Pain behavior was observed and rated before, during, and after CT removal. Pain behavior increased less in the topical EMLA group (mean ± SE, 4.4±0.39) compared with the IV morphine group (6.0±0.38; $p < 0.01$). No signs of infection were noted at the CT sites 24 or 48 h after CT removal. We conclude that EMLA cream is more effective than IV morphine in preventing the pain associated with CT removal. IMPLICATIONS: Postoperatively applying a topical anesthetic cream onto chest tube sites of chest surgery patients 3 h before chest tube removal is more effective than IV morphine in blunting pain response.

Effects of Different Modes of Delivery on Lung Volumes of Newborn Infants—Lee S, Hassan A, Ingram D, Milner AD. Pediatr Pulmonol 1999 May;27(5):318–321.

The aim of our study was to examine the effect of labor and birth canal compression on the rate of clearance of lung fluid. We recruited 10 babies born by vaginal delivery and 10 born by elective cesarean section (ECS). Thoracic gas volume (TGV) was measured using total body plethysmography, and functional residual capacity (FRC) by argon dilution, at ages 4–6 h and 24 h. Additional measurements were obtained at age 48 h in the infants born by ECS. Our results showed significant increases in mean TGV (from 23.1 mL/kg to 27.4 mL/kg, p = 0.002) and FRC (from 21.2 mL/kg to 24.1 mL/kg, p = 0.04) between 4–6 h and 24 h of age after ECS delivery. Lung volumes did not change significantly over the next 24 h (TGV, 27.4 mL/kg, p = 0.97; FRC, 25.5 mL/kg, p = 0.42). In those infants born by vaginal delivery, the mean TGV at 4–6 h and 24 h were 26.7 and 28.7 mL/kg, respectively (p = 0.09), and the mean FRC results were 23.1 and 24.9 mL/kg, respectively, p = 0.08). The TGV tended to be lower at 4–6 h in those born by ECS than in infants born vaginally (23.1 vs. 26.7 mL/kg, p = 0.05). We conclude that there is a delay of up to 24 h in the establishment of final lung volumes in babies born without exposure to labor or passage through the birth canal, and that this may explain the increased respiratory morbidity associated with delivery by ECS.

Medication Errors at the Administration Stage in an Intensive Care Unit—Tissot E, Cornette C, Demoly P, Jacquet M, Barale F, Capellier G. Intensive Care Med 1999 Apr; 25(4):353–359.

OBJECTIVE: To assess the type, frequency and potential clinical significance of medication-administration errors. DESIGN: Prospective study using the observation technique as described by the American Society of Health-System Pharmacists but eliminating the disguised aspect. SETTING: Medical intensive

KEYS TO EXCELLENCE IN THE CARE OF COPD PATIENTS

Health Tips Fact Sheets

These one-page fact sheets help you share your knowledge and expertise on important issues in respiratory health with the community. Pad of 50.
$7.50 ($10.00 nonmembers)

Topics Include:

Eating Right: Tips for the COPD Patient	**Item R25**
Exercising Safely with COPD	**Item R35**
Lessening the Effects of COPD	**Item R36**

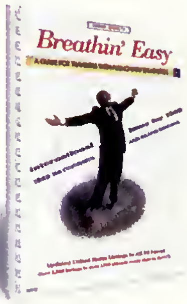
Quiz Sheets

Inpatients and outpatients alike will find these "IQ Tests" challenging and informative. These true-and-false tests feature detailed answers on a variety of subjects vital to respiratory care. Pad of 50.
$7.50 ($10.00 nonmembers)

Check Your COPD "IQ"	**Item R61**

Breathin' Easy Updated!

A Guide to Oxygen Refill Locations for Travelers with Pulmonary Disabilities



Helps your oxygen-dependent patients travel without worrying about oxygen refills. Look up your patient's destinations or the cities along their travel route. Listings include more than 3,000 oxygen refill locations in more than 2,000 cities in all 50 states. International listings are also included.
Item H10 $19.95

AUDIO CASSETTES

How to Implement a Pulmonary Rehabilitation Program

Describes all the steps and procedures necessary for the implementation of an effective pulmonary rehabilitation program. Presents ways to ensure all requirements are followed. Very comprehensive. By Julien M. Roy, BA, RRT.
Item PAD75 $15 (nonmembers $20)

A Business and Marketing Plan for Pulmonary Rehabilitation

Explains the business side of a pulmonary rehabilitation program. Describes how to write a business plan and how to market it. By Trina M. Limberg, BS, RRT.
Item PAD76 $15 (nonmembers $20)

Outcomes for Pulmonary Rehabilitation

Details all the requirements necessary to set up a basic outcomes measurement program for a pulmonary rehabilitation program. By Trina M. Limberg, BS, RRT.
Item PAD78 $15 (nonmembers $20)

INTERACTIVE CLINICAL SKILLS SOFTWARE

Chronic Obstructive Pulmonary Disease (COPD) Simulation

The user is asked to perform initial assessment and pulmonary function studies on a patient with chronic lung disease. Then, the user classifies the patient's disease as mild, moderate, severe restrictive, and/or obstructive disease. The simulation asks for home care and home equipment cleaning recommendations. Three months later the patient enters the ER with respiratory distress, requiring initial evaluation and therapy.
Item SP14 $65

IISPs

Ventilation/Perfusion Relationships in Health and Disease

Covers the different degrees of hypoxia commonly encountered in patients with pulmonary disease. Focuses on ventilation/ perfusion abnormalities, which account for much of the severe hypoxia in COPD, with alveolar hypoventilation also a contributing factor.
Item CS21 $12 (nonmembers $16)

VIDEOTAPES

Pulmonary Rehabilitation

Learn the components of a comprehensive pulmonary rehabilitation program, how to select appropriate candidates, and how to identify and set both short- and long-term patient goals. Also covers assessment of a patient's progress and follow-up. Featuring Barry M. Make, MD, and David J. Pierson, MD.
Item VC24 $49.95 (nonmembers $99)

To place your order, please call:

972-243-2272

Texas customers only, please add 8.25% sales tax (charged on product cost and shipping charges). Texas customers who are exempt from sales tax must supply an exemption certificate. MasterCard, Visa, Institutional Purchase Order or check payable to:

American Association for Respiratory Care
ATTN: Order Fulfillment Department
11030 Ables Lane • Dallas, Texas 75229
Phone 972-243-2272 Fax: 972-484-2720
www.aarc.org

Shipping Chart

$15 or Less	$4.25
$15.01 - $25.00	$5.25
$25.01 - $60.00	$7.25
$60.01 - $80.00	$9.25
$80.01 - $150.00	[illegible]
$150.01 - $250.00	$14.25
$251 or more	$16.25
International Orders Add $25	

care unit (ICU) in a university hospital. PATIENTS AND PARTICIPANTS: 2009 medication administration interventions by nurses. INTERVENTIONS: Pharmacist-performed observation of preparation and administration of medication by nurses, comparison with the original medical order and comparison with the data available in the literature. MEASUREMENTS AND RESULTS: 132 (6.6% of 2009 observed events) errors were detected. Their distribution is as follows: 41 dose errors, 29 wrong rate, 24 wrong preparation technique, 19 physicochemical incompatibility, 10 wrong administration technique and 9 wrong time errors. No fatal errors were observed, but 26 of 132 errors were potentially life-threatening and 55 potentially significant. CONCLUSION: According to this first observation-based study of medication administration errors in a European ICU, these errors were due to deficiencies in the overall organisation of the hospital medication track, in patient follow-up and in staff training.

Autopsy: Quality Assurance in the ICU—Gut AL, Ferreira AL, Montenegro MR. Intensive Care Med 1999 Apr;25(4):360–363.

OBJECTIVE: To examine the correlation between the clinical diagnosis and autopsy findings in adult patients who died in an intensive care unit (ICU). To determine the rate of agreement of the basic and terminal causes of death and the types of errors in order to improve quality control of future care. DESIGN: Retrospective study. SETTING: Adult ICU in a university hospital. PATIENTS: 30 adult patients who died in the ICU, with the exclusion of medicolegal cases. METHODS AND MAIN RESULTS: Anatomo-clinical meetings were held to analyze the pre- and postmortem correlations in 30 consecutive autopsies at the ICU of the University Hospital, School of Medicine of Botucatu/ UNESP, from January 1994 to January 1997. The rate of correct clinical diagnoses of the basic cause was 66.7%; in 23.3% of cases, if the correct diagnosis was made, management would have been different, as would have been the evolution of the patient's course (Class I error); in 10% of the cases the error would not have led to a change in management (Class II error). The rate of correct clinical diagnoses of terminal cause was 80%. CONCLUSIONS: The rate of recognition of the basic cause was 66.7%, which is consistent with the literature, but the Class I error rate was higher than that reported in the literature.

Right-to-Left Interatrial Shunt in ARDS: Dramatic Improvement in Prone Position—Legras A, Dequin PF, Hazouard E, Doucet O, Tranquart F, Perrotin D. Intensive Care Med 1999 Apr;25(4):412–414.

The mechanisms leading to shunting through a patent foramen ovale include high right-sided cardiac pressures and respiratory factors due to mechanical ventilation and also anatomical changes in the right atrium as described in the platypnea-orthodeoxia syndrome. We report a patient with the adult respiratory distress syndrome (ARDS) who had a right-to-left atrial shunt which decreased in the prone position, after which oxygenation improved. The patient was admitted to the intensive care unit because of ARDS due to an invasive fungal infection. He had a history of chronic lymphocytic leukemia and paradoxical embolisms through a patent foramen ovale. Despite mechanical ventilation and antifungal treatment he developed severe ARDS. He was therefore turned to the prone position. Blood gas values improved dramatically (arterial oxygen tension/fractional inspired oxygen ratio increasing from 59 to 278 torr). Transcranial Doppler sonography was performed with bubble study, which confirmed a massive right-to-left shunt in the supine position and which instantaneously decreased in the prone position. This case suggests that a decrease in right-to-left shunt in patients who have a patent foramen ovale could partly explain the improvement in hypoxemia in the prone position.

Mass Envenomations by Honey Bees and Wasps—Vetter RS, Visscher PK, Camazine S. West J Med 1999 Apr;170(4):223–227.

Stinging events involving honey bees and wasps are rare; most deaths or clinically important incidents involve very few stings (< 10) and anaphylactic shock. However, mass stinging events can prove life-threatening via the toxic action of the venom when injected in large amounts. With the advent of the Africanized honey bee in the southwestern United States and its potential for further spread, mass envenomation incidents will increase. Here we review the literature on mass stinging events involving honey bees and wasps (i.e., yellowjackets, wasps, and hornets). Despite different venom composition in the two insect groups, both may cause systemic damage and involve hemolysis, rhabdomyolysis, and acute renal failure. Victim death may occur due to renal failure or cardiac complications. With supportive care, however, most victims should be able to survive attacks from hundreds of wasps or approximately 1000 honey bees.

Formal Swallowing Evaluation and Therapy After Traumatic Brain Injury Improves Dysphagia Outcomes—Schurr MJ, Ebner KA, Maser AL, Sperling KB, Helgerson RB, Harms B. J Trauma 1999 May;46(5):817–821; discussion 821–823.

BACKGROUND: The incidence of swallowing dysfunction after brain injury is unknown. The efficacy of dysphagia therapy is also unknown. We reviewed our experience to define the incidence of swallowing dysfunction and efficacy of therapeutic intervention. METHODS: Patients with brain injury sustained between January of 1996 and December of 1997 were reviewed. All were screened with trials of oral intake. Abnormal findings were confirmed with a videofluoroscopic swallow study. Standard therapies included diet, posture, and behavior modifications. RESULTS: A total of 47 patients were evaluated. Bedside evaluations were normal in 14 patients, 2 patients had overt aspiration and underwent gastrostomy, and 31 patients were referred for a videofluoroscopic swallow study (66%). The videofluoroscopic swallow study was abnormal in 22 of 31 patients (71%). Of these, 4 additional patients required gastrostomy, 13 patients had laryngeal penetration or minor aspiration responsive to dysphagia therapy and were fed. Five other patients had silent aspiration and were fed by means of nasogastric tube; these five patients responded to dysphagia therapy and were able to resume oral intake. CONCLUSION: Dysphagia is common after severe head injury. With formal swallowing service intervention, aspiration is avoided. Therapeutic interventions can be used to restore oral intake.

Pulmonary Contusions: Quantifying the Lesions on Chest X-Ray Films and the Factors Affecting Prognosis—Tyburski JG, Collinge JD, Wilson RF, Eachempati SR. J Trauma 1999 May;46(5):833–838.

OBJECTIVES: To quantify pulmonary contusions on chest x-ray film and to evaluate factors correlating with the size of the pulmonary contusions, changes in the first 24 hours, the need for ventilatory assistance, and death. METHODS: The medical records and chest x-ray films of 103 patients with blunt chest trauma diagnosed as having a pulmonary contusion were reviewed. RESULTS: A pulmonary contusion score was developed (3 = one third of a lung; 9 = an entire lung). In the emergency department, pulmonary contusions were not present in 11, were mild (one ninth to two ninths of a lung) in 15 patients, moderate-severe (three ninths to nine ninths of a lung) in 53 patients, and very severe in 24 patients. Within 24 hours, the pulmonary contusion score increased in 26 patients by 7.9 ± 5.5 (SD). The 26 patients with an increasing contusion had a higher mortality rate (38% vs. 17%) ($p = 0.044$) and tended to need ventilatory assistance more frequently (73% vs. 49%) ($p = 0.061$). The 35 patients with very severe pulmonary contusions (pulmonary contusion score = 10–18) had the lowest P_{aO_2}:F_{IO_2} ratio at 24 hours (175 ± 103 mm Hg), longest hospital length of stay (28 ± 35 days), and the highest Injury Severity Score (26 ± 9). The factors correlating highest with a need for ventilatory support (57/103) were the 24 hour or initial P_{aO_2}/F_{IO_2} ratio < 300, an Injury Severity Score ≥ 24, Revised Trauma Score < 6.4, Glasgow Coma Scale score ≤ 12, and shock or need for blood in the first 24 hours ($p < 0.001$). Death correlated highly with a need for ventilatory assistance, Injury Severity Score ≥ 26, Revised Trauma Score ≤ 6.3, and Glasgow Coma Scale score ≤ 11 ($p < 0.001$). CONCLUSION: Quantifying and noting changes in the extent of the pulmonary contusions and P_{aO_2}/F_{IO_2} ratio during the first 24 hours may be of value in determining the need for ventilatory assistance and predicting outcome.

Pulmonary Gas Exchange During Intramedullary Fixation of Femoral Shaft Fractures—Weresh MJ, Stover MD, Bosse MJ, Jeray K, Kellam JF, Sims SH, Kline JA. J Trauma 1999 May;46(5):863–868.

BACKGROUND: This study was undertaken to determine if the alveolar dead space fraction (V_D/V_T) or the alveolar-arterial oxygen gradient (A-a DO_2) increased during intramedullary fixation of femoral shaft fractures. METHODS: Fifty hemodynamically stable patients with femur fractures were prospectively enrolled. Three serial measurements of V_D/V_T and A-a DO_2 were obtained immediately before femoral nailing (Pre), 30 minutes after nailing (+30), and 120 minutes after nailing (+120). V_D/V_T was determined by simultaneously measuring P_{aCO_2} and the steady-state end-tidal CO_2 ($PetCO_2$), where $V_D/V_T = (1 - PetCO_2/P_{aCO_2})$. RESULTS: V_D/V_T before nailing was 0.09 ± 0.09 (mean ± SD); at +30 and +120, V_D/V_T was 0.10 ± 0.06 and 0.08 ± 0.07, respectively ($p > 0.2$; paired t test, both time points). A-a DO_2 before nailing was 84 ± 85 mm Hg, and it did not change significantly at +30 (89 ± 69 mm Hg; $p = 0.51$ vs. Pre; paired *t*-test) or at +120 (51 ± 45 mm Hg). No difference in data was found with analysis by fracture classification or number of reamer passes. V_D/V_T and A-a DO_2, however, were both significantly increased in patients with lung contusion ($n = 6$) before nailing, but neither measurement increased after nailing. One patient developed fat embolism (V_D/V_T of 0.35 at Pre and 0.31 at +120), and another patient experienced postoperative pulmonary thromboembolism (V_DV_T increased from 0.06 at Pre to 0.17 at +120). CONCLUSION: The process of femoral nailing does not cause enough pulmonary embolization to alter pulmonary gas exchange as measured by V_D/V_T and A-a DO_2. If V_D/V_T is increased preoperatively, the likelihood of subsequent pulmonary dysfunction secondary to either preoperative lung injury or fat embolism is increased.

Respiratory Energetics During Exercise at High Altitude—Cibella F, Cuttitta G, Romano S, Grassi B, Bonsignore G, Milic-Emili J. J Appl Physiol 1999 Jun;86(6):1785-1792.

The purpose of this study was to assess the effect of high altitude (HA) on work of breathing and external work capacity. On the basis of

Y2K

Ventilator Strategy

How can you protect your ventilator dependent patients if the electricity and microprocessors shut down in the year 2000? Use VORTRAN Medical's new RespirTech PRO™ as your Y2K ventilator backup strategy.

ISSUES

- Y2K may adversely affect ventilator dependent patients.
- Rental equipment may not be available.
- Existing equipment may need recalibrating.

SOLUTION

The RespirTech PRO is a fully automatic, gas powered, single patient use resuscitator which can be used as a backup ventilator. It requires a continuous flow of gas and is **NOT DEPENDENT ON COMPUTERS OR ELECTRICITY**. Its compact size and weight make it easy to store and use, and it only costs $29 each.

CONCLUSION

The RespirTech PRO can serve as an optimal Y2K backup for ventilator problems. It is MRI compatible and is also an excellent transport ventilator. The easy to use RespirTech PRO allows hands free ventilation during emergency situations both in and out of the hospital.

Call (800) 434-4034 or visit our website at http:\\www.vortran.com.

VORTRAN Medical Technology 1, Inc.
3941 J Street, Suite 354
Sacramento, CA 95819-3633
Tel: (916) 733-6870 • (800) 434-4034
Fax: (916) 454-0490 • E-mail: *office@vortan.com*

Circle 112 on reader service card

simultaneous records of esophageal pressure and lung volume, the mechanical power of breathing (Wrs) was measured in four normal subjects during exercise at sea level (SL) and after a 1-mo sojourn at 5,050 m. Maximal exercise ventilation ($\dot{V}_{Emax}$) and maximal Wrs were higher at HA than at SL (mean 185 vs. 101 L/min and 129 vs. 40 cal/min, respectively), whereas maximal O_2 uptake averaged 2.07 and 3.03 L/min, respectively. In three subjects, the relationship of Wrs to minute ventilation ($\dot{V}_E$) was the same at SL and HA, whereas, in one individual, Wrs for any given $\dot{V}_E$ was consistently lower at HA. Assuming a mechanical efficiency (E) of 5%, the O_2 cost of breathing at HA and SL should amount to 26 and 5.5% of maximal O_2 uptake, whereas for E of 20% the corresponding values were 6.5 and 1.4%, respectively. Thus, at HA, Wrs may substantially limit external work unless E is high. Although at SL $\dot{V}_{Emax}$ did not exceed the critical $\dot{V}_E$, at which any increase in $\dot{V}_E$ is not useful in terms of body energetics even for E of 5%, at HA $\dot{V}_{Emax}$ exceeded critical $\dot{V}_E$ even for E of 20%.

Posture Effects on Timing of Abdominal Muscle Activity During Stimulated Ventilation—Abe T, Yamada T, Tomita T, Easton PA. J Appl Physiol 1999 Jun;86(6):1994-2000.

In humans during stimulated ventilation, substantial abdominal muscle activity extends into the following inspiration as postexpiratory expiratory activity (PEEA) and commences again during late inspiration as preexpiratory expiratory activity (PREA). We hypothesized that the timing of PEEA and PREA would be changed systematically by posture. Fine-wire electrodes were inserted into the rectus abdominis, external oblique, internal oblique, and transversus abdominis in nine awake subjects. Airflow, end-tidal CO_2, and moving average electromyogram (EMG) signals were recorded during resting and CO_2-stimulated ventilation in both supine and standing postures. Phasic expiratory EMG activity (tidal EMG) of the four abdominal muscles at any level of CO_2 stimulation was greater while standing. Abdominal muscle activities during inspiration, PEEA, and PREA, were observed with CO_2 stimulation, both supine and standing. Change in posture had a significant effect on intrabreath timing of expiratory muscle activation at any level of CO_2 stimulation. The transversus abdominis showed a significant increase in PEEA and a significant decrease in PREA while subjects were standing; similar changes were seen in the internal oblique. We conclude that changes in posture are associated with significant changes in phasic expiratory activity of the four abdominal muscles, with systematic changes in the timing of abdominal muscle activity during early and late inspiration.

Neuromechanical Interaction in Human Snoring and Upper Airway Obstruction—Huang L, Williams JE. J Appl Physiol 1999 Jun;86(6):1759-1763.

The fact that snoring and obstructive apnea only occur during sleep means that effective neuromuscular functioning of the upper airway during sleep is vital for the maintenance of unimpeded breathing. Recent clinical studies in humans have obtained evidence demonstrating that upper airway neural receptors sense the negative pressure generated by inspiration and 'trigger,' with a certain delay, reflex muscle activation to sustain the airway that might otherwise collapse. These findings have enabled us to propose a model in which the mechanics is coupled to the neuromuscular physiology through the generation of reflex wall stiffening proportional to the retarded fluid pressure. Preliminary results on this model exhibit three kinds of behavior typical of unimpeded breathing, snoring, and obstructive sleep apnea, respectively. We suggest that the increased latency of the reflex muscle activation in sleep, together with the reduced strength of the reflex, have important clinical consequences.

Does Treatment of Asthmatic Children with Inhaled Corticosteroids Affect Their Adult Height?—Van Bever HP, Desager KN, Lijssens N, Weyler JJ, Du Caju MV. Pediatr Pulmonol 1999 Jun;27(6):369-375.

In this retrospective study, adult height was assessed in young adult asthmatics who were treated with inhaled corticosteroids (ICs) during childhood (n = 42; 26 boys) and compared to those obtained in asthmatic patients who were never treated with ICs during childhood (n = 43; 23 boys). Standing height of all subjects and their parents was measured. Height data were analyzed using actual length and target height in centimeters, standard deviation scores (SDS), and difference between adult height of the patients and their target height (adult height minus target height). Mean adult height was the same in subjects who took ICs during childhood as compared to those who had never received ICs (boys: 179.3cm± 6.8 vs. 180.4 cm ± 5.6; girls: 165.8 cm± 7.5 vs. 167.7 cm ± 7.2). SDS of adult height was also not different between the two groups: in subjects who did not take ICs it was 0.89 ± 1.00, while in those who took ICs it was 0.66 ± 1.10 (p= 0.31). SDS of target height was also not different between the two groups: in subjects not taking ICs it was 0.95 ± 0.86, while in those who took ICs it was 0.28 ± 0.76 (p= 0.30). However, subjects who took ICs during childhood showed a statistically significant lower value of adult height minus target height than those who never took ICs (whole group: -0.003 ± 5.9 vs. 2.54 ± 4.8, p = 0.03 ; boys: 0.004 ± 5.8 vs. 3.09 ± 4.5, p = 0.04 ; girls: -0.075± 6.3 vs. 1.91 ± 5.2, p= 0.31). Patients on ICs during childhood who had ever been hospitalized for asthma showed a lower value for adult height minus target height than those who took ICs but were never hospitalized (-3.08 ± 7.8 vs. 1.06 ± 4.8, p = 0.046). A logistic regression analysis predicting growth impairment showed that the best-fitting model was one that used only ICs as a dependent variable (crude odds ratio, 3.3; 95% CI, 1.3-8.4). Patients who were treated with ICs in combination with intranasal corticosteroids (treatment for rhinitis) tended to have a lower value of adult height minus target height than the other children, but the difference was not statistically significant (p = 0.07). We conclude that although adult height was the same in young adults who were treated with ICs during childhood compared to those who were not treated with ICs during childhood, there was a statistically significant difference between the two groups for adult height minus target height, suggesting mild growth retardation in patients who took ICs during childhood. These findings may be explained by the use of ICs, but it seems more likely that a difference in asthma severity between both groups was responsible for it.

Early Prediction of Neonatal Chronic Lung Disease: A Comparison of Three Scoring Methods—Yoder BA, Anwar MU, Clark RH. Pediatr Pulmonol 1999 Jun;27(6):388-394.

A variety of postnatal therapies have been and will be evaluated for prevention or treatment of neonatal chronic lung disease (CLD). A simple method for early selection of the highest risk infants would optimize intervention trials. Our study compared a clinical scoring system for predicting neonatal CLD (defined at 36 weeks postconceptional age) with previous regression models developed by Sinkin et al. (Sinkin model) [Pediatrics 1990;86:728-736] and Ryan et al. (Ryan model) [Eur J Pediatr 1996;668-671] in two distinct populations. A respiratory failure score (RFS) was prospectively developed for infants at < 32 weeks of gestation admitted to the Wilford Hall Medical Center from January 1990-December 1992. Logistic regression modeling identified three independent predictors of CLD: gestation, birth weight, and RFS. Applying a modified RFS score (to include gestation and birth weight), the RFS, Sinkin, and Ryan models were compared among high-risk infants admitted to Wilford Hall from January 1993-December 1995, and to Crawford Long Hospital (Atlanta, GA) from January 1993-December 1994. Predictive values, sensitivity, specificity, and receiver operating characteristic (ROC) curves were determined for the primary outcome variable: CLD at 36 weeks of corrected gestation. Of 248 infants at <32 weeks admitted to Wilford Hall, 220 survived >7 days. Thirty of 31 (97%) infants diagnosed with CLD were <29 weeks or ≤1,000 g at birth. Despite important demographic and treatment differ-

INOvent® delivery system

FOR NITRIC OXIDE THERAPY

Seamless, Integrated and Complete

The INOvent delivery system for superior delivery and monitoring of inhaled Nitric Oxide

The INOvent delivery system is unparalleled for flexibility, accuracy and ease of use.

Flexibility gives you efficiency

- The INOvent delivery system is flexible and portable – use it with most mechanical ventilators and ventilator modes.

Accuracy gives you enhanced patient protection

- The INOvent delivery system provides a constant concentration of user-set NO to the patient, while continuously monitoring inspired NO, NO_2, and O_2.
- It includes a comprehensive alarm system, dual cylinder connection, 30-minute battery backup, and integrated manual NO delivery capability.

Ease of use gives you extra convenience

The user interface includes:

- A large, easy-to-read display of set and measure values
- Direct setting of NO without calculation
- Simple calibration.

Combine flexibility, accuracy, and ease of use and you get dependable performance day in and day out – that's the INOvent delivery system.

Find out more. Call Datex-Ohmeda – 800-345-2700 or 608-221-1551

Circle 129 on reader service card

Datex Ohmeda

INOvent delivery system, 510K pending in U.S.

ences between the study populations, similar ROC curves were found for each scoring method when individually evaluated among the three study groups. The RFS method at 72 h demonstrated the greatest area under the ROC curve for prediction of neonatal CLD in the groups as a whole. Application of the RFS method for early prediction of neonatal CLD at age 72 h should improve patient selection for early prevention trials.

Clinical Features of Obstructive Sleep Apnea Hypoventilation Syndrome in Otherwise Healthy Children—Rosen CL. Pediatr Pulmonol 1999 Jun;27(6):403-409.

Obstructive sleep apnea hypoventilation syndrome (OSAHS) is an important public health problem. However, major gaps exist in our knowledge about the clinical features of this disorder in the pediatric age group. The purpose of this study was to examine clinical features of OSAHS diagnosed by polysomnography in otherwise healthy children. In this cross-sectional study, 326 children without underlying medical conditions (5.8 ± 3.0 years, range 1-12 years; 56% male) were recruited from patients referred by primary care and otorhinolaryngology physicians for evaluation of snoring and difficulty breathing. Ethnic group distribution was African-American (38%), Caucasian (30%), and Hispanic (31%). Complaints of daytime tiredness or sleepiness were reported in 29% of the children. All children underwent overnight polysomnography (N = 330 studies). OSAHS was diagnosed in 59% of the children, based on polysomnographic criteria. The remaining children had either primary snoring (25%); no snoring (10%), or upper airway resistance syndrome (6%). Neither male gender nor obesity increased the likelihood for the diagnosis of OSAHS. However, the incidence of obesity in the study population (28%) was more than twice that of the general pediatric population. African-American children had a greater likelihood for OSAHS diagnosis compared to Hispanic or Caucasian children. Daytime complaints of sleepiness or tiredness were not more common in children diagnosed with OSAHS than in the children without OSAHS. As expected, tonsillar hypertrophy increased the likelihood of OSAHS diagnosis. In summary, many of the clinical features of childhood OSAHS are in marked contrast to those in adults.

Effect of Single versus Multiple Dosing on Perfluorochemical Distribution and Elimination During Partial Liquid Ventilation—Miller TF, Milestone B, Stern R, Shaffer TH, Wolfson MR. Pediatr Pulmonol 1999 Jun;27(6): 410-418.

The objective of this study was to quantitate perfluorochemical (PFC) elimination kinetics during partial liquid ventilation (PLV) following an initial fill with or without hourly dosing. Young New Zealand rabbits were studied in two groups: Gr I (n = 6), PLV with a single dose of PFC liquid (perflubron: LiquiVent, Alliance Pharmaceutical Corp.); and Gr II (n = 5), PLV with PFC liquid and multiple hourly dosing . All rabbits were studied for 4 h, following initial instillation of a volume of PFC liquid equal to the measured gas functional residual capacity. Animals were ventilated at a constant breathing frequency (30 br/min), tidal volume (9.3 ± 0.3 SE mL/kg), positive end expiratory pressure (4 cm H_2O), and inspiratory time (0.30 s). PFC saturation of mixed expired gas (PFC-Sat) was assessed with a thermal conductivity analyzer, and PFC elimination was calculated from PFC-Sat, minute ventilation, and temperature of the expired gas. In GR II, PFC was supplemented hourly at a volume determined by PFC elimination calculations. The results demonstrated a decrease in PFC-sat and PFC loss with time, independent of group ($p < 0.05$). In addition, with hourly supplementation (GR II), PFC-Sat and PFC elimination over time was significantly ($p < 0.05$) greater than in animals (GR I) which did not receive additional doses. These data demonstrate that the PFC elimination rate is not constant and is related to the amount of PFC in the respiratory system. This may have occurred due to distributional differences of ventilation and PFC liquid between the single and multiple dosing groups. These findings also suggest that evaluation of PFC concentrations in expired gas may be a clinically useful index of intrapulmonary PFC distribution during PLV, and that maintained elevation of expired gas PFC saturation may guide optimal PFC dosing intervals and distribution to maximize protection against barotrauma.

Lipid-Laden Macrophages in Bronchoalveolar Lavage Fluid as a Marker for Pulmonary Aspiration—Knauer-Fischer S, Ratjen F. Pediatr Pulmonol 1999 Jun;27(6):419-422.

An increased lipid content in alveolar macrophages of bronchoalveolar lavage (BAL) fluid is thought to be a useful indicator for recurrent pulmonary aspiration. To assess whether pulmonary diseases unrelated to aspiration can raise the lipid content in alveolar macrophages, we evaluated Oil-Red-O-stained smears of BAL fluid in 18 children aged 3-15 years undergoing elective surgery for nonpulmonary illnesses under general anesthesia and in 18 children aged 1-16 years who had pulmonary diseases without clinical evidence of aspiration (pneumonia, exogenous allergic alveolitis, or cystic fibrosis). A semiquantitative lipid-laden macrophage (LLM) index was determined for each patient. LLM indices in children without pulmonary disease were higher than those published for healthy adults. In children with pulmonary diseases but without evidence of aspiration, a significantly higher LLM index was observed compared to controls. The LLM indices of children with pulmonary diseases were similar to those published by other authors for children with pulmonary aspiration. We conclude that an elevated LLM index in alveolar macrophages of BAL can be found in a variety of pulmonary diseases in which there is no clinical evidence of aspiration and is therefore unlikely to be a specific parameter for silent pulmonary aspiration.

Relation Between Pulse Oximetry and Clinical Score in Children with Acute Wheezing Less Than 24 Months of Age—Pavon D, Castro-Rodriguez JA, Rubilar L, Girardi G. Pediatr Pulmonol 1999 Jun;27(6):423-427.

The aim of this study was to determine the relation between transcutaneous hemoglobin oxygen saturation, measured by pulse oximetry (S_{pO_2}), and clinical score values in 138 infants (mean ± SD, 6.6 ± 5.5 months of age) with acute wheezing episodes presenting in a primary care outpatient setting. A single investigator evaluated the severity of the acute wheezing episodes by assigning a clinical score and was unaware of the S_{pO_2} values. Another investigator measured S_{pO_2} values on all subjects. The mean (± SD) S_{pO_2} value was 98.2 ± 1.1% for children with clinical scores of 2-5 (n = 32); 95.41± 1.5% for those with scores of 6-7 (n = 82), and 92.9 ± 2% for children with scores of 8-10 (n = 24), ($p < 0.001$ by Bonferroni's multiple comparison, when all two-way comparisons were done for each pair of results). The clinical score showed a good correlation with S_{pO_2} (r = -0.76; 95% CI, -0.83 to -0.68). We conclude that if pulse oximetry is not available, it is advisable to include oxygen in the therapy of wheezy infants with clinical scores values > 8.

The Efficacy of Exercise as an Aid for Smoking Cessation in Women: A Randomized Controlled Trial—Marcus BH, Albrecht AE, King TK, Parisi AF, Pinto BM, Roberts M, Niaura RS, Abrams DB. Arch Intern Med 1999 Jun 14;159(11):1229-1234.

BACKGROUND: Smoking prevalence rates among women are declining at a slower rate than among men. OBJECTIVE: To determine if exercise, a healthful alternative to smoking, enhances the achievement and maintenance of smoking cessation. METHODS: Two hundred eighty-one healthy, sedentary female smokers were randomly assigned to either a cognitive-behavioral smoking cessation program with vigorous exercise (exercise) or to the same program with equal staff contact time (control). Subjects participated in a 12-session, group-based smoking cessation program. Additionally, exercise subjects were required to attend 3 supervised exercise sessions per week and control subjects were required to participate in 3 supervised health education lectures per week. Ab-

HONE YOUR OXYGENATION KNOWLEDGE WITH PROFESSIONAL EDUCATION PRODUCTS FROM THE AARC

Monitoring Oxygenation of the Critically Ill Patient

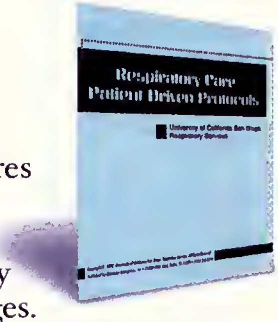
Reviews the determinants of tissue oxygenation in the critically ill patient and the techniques available for assessment. Also discusses "supply dependency" of tissue oxygen utilization in ARDS and how it affects management. Featuring Leonard D. Hudson, MD, and David J. Pierson, MD.
Item VC34 $49.95 (nonmembers $99)

Unconventional Methods for Adult Oxygenation and Ventilation Support

Provides an overview of new and experimental techniques for adult oxygenation and ventilation support. Discusses the techniques, theoretical rationales, methods of application, and experimental evidence of the effectiveness of these unconventional methods. Featuring James K. Stoller, MD, and David J. Pierson, MD.
Item VC36 $49.95 (nonmembers $99)

Respiratory Care Patient-Driven Protocols

Written by UCSD faculty, one of the country's leading institutions in the development of protocols. This book serves as an excellent resource in the processes of developing, implementing, or refining care plans. Features 25 complete protocols with an introduction on how to implement PDPs in your facility. Authors include Jan Phillips-Clar, BS, RRT; Richard Ford, BS, RRT; Timothy Morris, MD; and David Burns, MD. Paperback. 205 pages. Published in 1998.
Item PA801 $85.00 ($99.00 nonmembers)



Basic Oxygen Administration

Presents the primary goals of oxygen therapy and the clinical conditions that warrant oxygen administration. Also covers oxygen sampling techniques for various types of patients including newborns and various oxygen administration appliances. Includes oxygen administration hazards, assessment factors for evaluation of oxygen delivery, the principles of humidification and factors affecting efficiency of devices, proper attachment of oxygen devices, and trouble-shooting of bubble humidifiers.
Item PEL3 $65

To place your order, please call:

972-243-2272

Texas customers only, please add 8.25% sales tax (charged on product cost and shipping charges). Texas customers who are exempt from sales tax must supply an exemption certificate. MasterCard, Visa, Institutional Purchase Order or check payable to:

American Association for Respiratory Care

ATTN: Order Fulfillment Department
11030 Ables Lane • Dallas, Texas 75229
Phone 972-243-2272 Fax: 972-484-2720

www.aarc.org

Shipping Chart	
$25.01 - $60.00	$7.25
$60.01 - $80.00	$8.25
$80.01 - $150.00	$10.25
$150.01 - $250.00	$14[illegible]
$251 or more	$16.25
International Orders Add $25	

stinence from smoking was based on self-report, was verified by saliva cotinine level, and was measured at 1 week after quit day (week 5), end of treatment (week 12), and 3 and 12 months later (20 and 60 weeks after quit day, respectively). RESULTS: Compared with control subjects (n = 147), exercise subjects (n = 134) achieved significantly higher levels of continuous abstinence at the end of treatment (19.4% vs 10.2%, p = 0.03) and 3 months (16.4% vs 8.2%, p=0.03) and 12 months (11.9% vs 5.4%, p=0.05) following treatment. Exercise subjects had significantly increased functional capacity (estimated $\dot{V}_{O_2}$ peak, 25 ± 6 to 28 ± 6, $p<0.01$) and had gained less weight by the end of treatment (3.05 vs 5.40 kg, p = 0.03). CONCLUSIONS: Vigorous exercise facilitates short- and longer-term smoking cessation in women when combined with a cognitive-behavioral smoking cessation program. Vigorous exercise improves exercise capacity and delays weight gain following smoking cessation.

Sex Differences Among Adults Presenting to the Emergency Department with Acute Asthma. Multicenter Asthma Research Collaboration Investigators—Singh AK, Cydulka RK, Stahmer SA, Woodruff PG, Camargo CA Jr. Arch Intern Med 1999 Jun 14;159(11):1237-1243.

BACKGROUND: Asthma is an increasing problem worldwide, particularly among women. Sex differences in acute asthma presentation, management, or outcome would have important medical and economic implications. OBJECTIVE: To compare emergency department (ED) visits for acute asthma among women vs men. METHODS: We performed a prospective cohort study as part of the Multicenter Asthma Research Collaboration. Patients in the ED, aged 18 to 54 years, who presented with acute asthma underwent a structured interview in the ED and another by telephone 2 weeks later. The study was performed at 36 EDs in 18 states. Pregnant women with asthma were excluded (n=53). RESULTS: Of 1228 patients, 64.3% were women. Women did not differ significantly from men by age or education level, but women were more likely to be insured, have a primary care provider, and use inhaled corticosteroids. Women had a higher mean ±SD peak expiratory flow rate than men, both early (expressed as percent predicted) (53%±21% vs 41%±18%; $p<0.001$) and late (77%±24% vs 65%±21%; $p<0.001$) in the ED stay. Despite this, women were more likely to be admitted to a hospital (multivariate odds ratio, 2.2; 95% confidence interval; 1.3-4.0) than men. At 2-week follow-up, women had not experienced more relapse events (odds ratio, 1.1) but were 1.5 times more likely to report an ongoing exacerbation (95% confidence interval; 1.0-2.4). CONCLUSIONS: Of adults who presented to the ED with acute asthma, women were almost twice as common as men. Although men received less outpatient care and had worse pulmonary function, women were more likely to be admitted to the hospital and to report an ongoing exacerbation at follow-up. Further studies are needed to better understand the complex relationship between sex and acute asthma.

Effect of Cigar Smoking on the Risk of Cardiovascular Disease, Chronic Obstructive Pulmonary Disease, and Cancer in Men—Iribarren C, Tekawa IS, Sidney S, Friedman GD. N Engl J Med 1999 Jun 10;340(23):1773-1780.

BACKGROUND: The sale of cigars in the United States has been increasing for the past six years. Cigar smoking is a known risk factor for certain cancers and for chronic obstructive pulmonary disease (COPD). However, unlike the relation between cigarette smoking and cardiovascular disease, the association between cigar smoking and cardiovascular disease has not been clearly established. METHODS: We performed a cohort study among 17,774 men 30 to 85 years of age at baseline (from 1964 through 1973) who were enrolled in the Kaiser Permanente health plan and who reported that they had never smoked cigarettes and did not currently smoke a pipe. Those who smoked cigars (1546 men) and those who did not (16,228) were followed from 1971 through the end of 1995 for a first hospitalization for or death from a major cardiovascular disease or COPD, and through the end of 1996 for a diagnosis of cancer. RESULTS: In multivariate analysis, cigar smokers, as compared with nonsmokers, were at higher risk for coronary heart disease (relative risk, 1.27; 95 percent confidence interval, 1.12 to 1.45), COPD (relative risk, 1.45; 95 percent confidence interval, 1.10 to 1.91), and cancers of the upper aerodigestive tract (relative risk, 2.02; 95 percent confidence interval, 1.01 to 4.06) and lung (relative risk, 2.14; 95 percent confidence interval, 1.12 to 4.11), with evidence of dose-response effects. There appeared to be a synergistic relation between cigar smoking and alcohol consumption with respect to the risk of oropharyngeal cancers and cancers of the upper aerodigestive tract. CONCLUSIONS: Independently of other risk factors, regular cigar smoking can increase the risk of coronary heart disease, COPD, and cancers of the upper aerodigestive tract and lung. *See the related editorial: **Cigars and Public Health.** Satcher D. N Engl J Med 1999 Jun 10;340(23):1829-1831.*

Initial Serum Ferritin Levels in Patients with Multiple Trauma and the Subsequent Development of Acute Respiratory Distress Syndrome—Sharkey RA, Donnelly SC, Connelly KG, Robertson CE, Haslett C, Repine JE. Am J Respir Crit Care Med 1999 May;159(5 Pt 1):1506-1509.

Acute respiratory distress syndrome (ARDS) represents a catastrophic form of inflammatory lung injury that occurs unpredictably in some, but not all, at-risk patients. In this study, we investigated serum ferritin as a marker for ARDS development in a homogenous group of patients at-risk because of multiple trauma. We hypothesized that since ferritin synthesis is increased by proinflammatory cytokines, which are increased and implicated in ARDS, that ferritin levels would increase and that ferritin increases would correlate with the degree of inflammation and therefore the development of ARDS. We studied 42 patients (25 male, 17 female) who as a consequence of multiple trauma became at-risk for developing ARDS. Using the European/American Consensus definition for ARDS, 16 (38%) patients subsequently developed ARDS (11 male and five female). We found that initial serum ferritin levels correlated with the subsequent development of both ARDS (progression to ARDS, median = 638 ng/mL; (range, 70 to 4,500) versus nonprogression to ARDS = 185 ng/mL; range, 12 to 2,850) (p = 0.02, r = -0.27) and multiple organ failure ($p < 0.05$, r = 0.39). Using our previously established cutoff points for serum ferritin, the positive predictive value was 62% for men and 75% for women. Initial serum ferritin levels also correlated with a measurement of the degree of initial trauma injury, i.e., the injury severity score (ISS) ($p < 0.05$, r = 0.37). However, there was no correlation between serum ferritin levels and other markers of clinical injury, namely, lowest P_{aO_2}/F_{IO_2} ratio (p = 0.67), days requiring ventilation (p = 0.09), or mortality (p = 0.42). A significant association existed between serum ferritin levels and products of endothelial activation, i.e., sE-selectin ($p < 0.04$, r = 0.37) and sICAM-1 ($p < 0.01$, r = 0.21). In the future, with the development of novel anti-inflammatory therapies, early identification of specific high-risk patients would allow the institution of these therapies and thereby increase the chances of reducing ARDS morbidity and mortality.

Respiratory Therapists and Conscious Sedation

The ability to sense pain is important for survival, because pain is a warning to protect an injured area against further damage. Pain triggers autonomic responses that have widespread effects on metabolism, immune function, and the cardiovascular system. The stress produced by pain can lead to hypertension, tachycardia, myocardial ischemia, and myocardial infarction. Pain is a subjective, powerful, unpleasant experience and is imbued with strong emotional significance, because it can mean loss of function, disintegration of physical appearance, suffering, and death. It is difficult to measure pain objectively, and individual responses to the same degree of injury vary widely. Fear and pain are frequent adverse effects of many clinical interventions, and both the physical and emotional components of the pain experience associated with clinical interventions can increase morbidity and mortality. Thus, minimizing fear and pain is an important part of clinical care.

Many approaches are available to minimize the unpleasant aspect of diagnostic and therapeutic procedures. Patient preparation, education, relaxation exercises, hypnosis, and drugs may be useful. Conscious sedation (CS) refers to the pharmacological mitigation of pain and fear during medical procedures. Improved patient comfort and outcome are the goals of sedation, and, ideally, sedated patients should remain conscious and able to communicate, to protect their own airway, and to breath adequately. However, because of variations in patient responses, consciousness may be lost during sedation.

Institutional standards for safe and effective provision of CS are required by the Joint Commission for Accreditation of Health Care Organizations, and other regulatory agencies. These standards must be adhered to throughout the institution, whether sedation is provided by a nurse, anesthesiologist, or other health care provider. Many concerned groups have developed guidelines for providing safe CS. Respiratory therapists should understand sedative and analgesic pharmacology and actively participate in provision of CS.[1] Since most of the complications of CS relate to airway compromise, respiratory therapists are uniquely qualified to safeguard patients and improve outcomes during CS.

Most guidelines and many clinical reports differentiate several levels of sedation. Often a distinction is drawn between "conscious" and "deep" sedation.[2] However, the progression from "conscious sedation" to "deep sedation" to "general anesthesia" is often difficult to control clinically, and each level involves increased risks and requires more intensive monitoring and support. Table 1 shows the definitions of these states and provides suggested requirements for monitoring.

All published standards insist on the presence of more than one health care provider during the period of sedation (at least the operator and a monitoring assistant). The American Dental Society of Anesthesiology Guidelines suggest that deep sedation and general anesthesia are indistinguishable and that at least 3 qualified people must be continually present during the sedation period.[3] These standards also suggest that one person must have as their *sole responsibility* continual patient monitoring and recording of vital signs. The guidelines suggest that the additional individual be an "anesthesia assistant, anesthesia technician, nurse, physician (anesthesiologist), or dentist." Other sections of these guidelines deal with the need to continually assess and assure oxygenation, ventilation, and temperature maintenance. Although suggestions are offered as to how to monitor these vital functions, the decision of whether to use a particular device and the frequency of observations are left to the responsible clinician.[4] What is not left to the discretion of the dentist is the number of personnel necessary and the fact that they must be specially qualified and assigned to monitor *only* a single patient's vital functions and progress of sedation. There must be resuscitation equipment immediately available and individuals trained to use it. Dentists are required to be competent at providing CS as part of advanced dental practice. A proposed curriculum includes a minimum of 60 hours of instruction and 100 hours of supervised practice in CS.[5]

The Association of Operating Room Nurses' proposed standards for monitoring patients receiving intravenous CS[6] include the requirement that the person managing the care of the patient "should have no other responsibilities that would leave the patient unattended or compromise continuous monitoring." The need for specially qualified personnel is implied by the requirement that the nurse be able to provide airway management skills and "may be required to be ACLS (Advanced Cardiac Life Support) certified." Resuscitation equipment is explicitly required. Requirements for monitoring and the frequency of recording of vital signs are referenced to certified nurse anesthetist materials.

Table 1. Monitoring Requirements for Various Levels of Sedation

Level of sedation	Definition	Suggested monitors
Conscious sedation	Minimally depressed level of consciousness. Patient retains ability to maintain airway independently and continuously and to respond to physical stimulation and verbal command.	• Dedicated monitoring assistant • Pulse oximetry • Intravenous access • Blood pressure measurement every 15 minutes
Deep sedation	Depressed consciousness accompanied by partial loss of protective reflexes and inability to respond purposefully to verbal command.	• Skilled airway person • Monitoring and recording person • Intravenous access • Pulse oximetry • Continuous electrocardiogram monitoring • Blood pressure measurement every 5 minutes
General anesthesia	Unconsciousness accompanied by partial or complete loss of protective reflexes and inability to maintain an airway independently.	• Anesthesia personnel • Anesthesia assistant • Intravenous access • Pulse oximetry • Carbon dioxide measurement device • Continuous electrocardiogram monitoring • Blood pressure measurement every 5 minutes • Other requirements dictated by patient's physiological condition

Conducting painful procedures with pediatric patients engenders considerable anxiety for caregivers. Because children are frequently uncooperative, sedation is often needed in order to perform procedures safely.[7,8] Various bodies have proposed monitoring standards for sedation of children.[9,10] The need for specific guidelines for evaluation, patient consent, monitoring, and collaboration with anesthesiology departments has been advocated.[11] Having a responsible individual overseeing the sedation and monitoring the patient is an essential part of these suggestions. These and other guidelines for sedation of children explicitly require that the person monitoring the child during sedation have the ability to treat airway and other emergencies.[12,13]

Gastrointestinal endoscopy is an uncomfortable procedure often performed under sedation,[14,15] and complications from the procedure and sedation are frequently reported. The British Society of Gastroenterology's standards for monitoring and sedation during and following endoscopy[16] require patient risk assessment (using the American Society of Anesthesiologists' patient classification system); that personnel have resuscitation skills; continuous monitoring; and specific monitoring equipment (eg, pulse oximetry) recommendations for high-risk patients.

The British Thoracic Society's Standards of Care Committee's guidelines for care during bronchoscopy require oxygenation monitoring (pulse oximetry), an intravenous line, resuscitation equipment, and personnel with resuscitation skills.[17] The use of anesthetic agents (eg, propofol) are specifically excluded from the usual practice, unless anesthesiology personnel are participating in the process.[18]

From the above summaries of existing CS guidelines, several common recommendations are apparent:

1. The person conducting the procedure cannot be the person monitoring and/or sedating the patient.
2. Intravenous access is the preferred means of administering sedative, because with this method the onset and cessation of the sedative effects are rapid and titration to the desired level is predictable.
3. The ability to establish and maintain an artificial airway must be available *immediately* whenever sedation is initiated. The inability to provide this level of expertise is a contraindication to CS.
4. Airway and resuscitation equipment *and* personnel with competence in using it must also be immediately available.

Who should participate in CS? The need for individuals with patient assessment skills, monitoring competencies, and airway skills could easily be filled by experienced respiratory therapists. The assistant role is often filled by a nurse, but a nurse might not have the required airway skills. Most experienced nurses can start intravenous infusions, administer intravenous medications, monitor patients, and assess the level of sedation. Can a respiratory therapist perform these tasks? With additional training and experience, respiratory therapists can become competent in inserting intravenous catheters and titrating analgesics and sedatives according to a physician's direction. Sedation scales based on the patient's responsiveness can be used to assess and record the progress of sedation.[19]

Only a few states currently allow respiratory therapists to perform all these functions, but most states allow them to perform many of these activities. Respiratory therapists often provide airway skills on an emergency basis to patients receiving CS, and they monitor and support patients on mechanical ventilation during procedures, so it is a logical extension that they should provide care during administration of CS. The ideal situation may be to have both a respiratory therapist and a nurse involved in CS procedures. However, financial considerations may limit the number of individuals involved in the procedure, and if only one additional person can be present, there are compelling reasons for employing a respiratory therapist as the sedation assistant. The American Association for Respiratory Care has endorsed the use of respiratory therapists for CS. Reference is made to "Practice guidelines for sedation and analgesia by nonanesthesiologists" for suggestions in the structure of training of practitioners for sedation and analgesia.[20] By developing expertise in this emerging field, respiratory therapists can improve the safety and quality of patient care.

Charles G Durbin Jr MD
Department of Anesthesiology
University of Virginia Health Science Center
Charlottesville, Virginia

REFERENCES

1. AARC. Administration of sedative and analgesic medications by respiratory care practitioners: a position statement from the American Association for Respiratory Care. Respir Care 1998;43(8):655.
2. Phero JC. Pharmacologic management of pain, anxiety, and behavior: conscious sedation, deep sedation, and general anesthesia. Pediatr Dent 1993;15(6):429–434.
3. Rosenberg MB, Campbell RL. Guidelines for intraoperative monitoring of dental patients undergoing conscious sedation, deep sedation, and general anesthesia. Oral Surg Oral Med Oral Pathol 1991; 71(1):2–8.
4. Matthews RW, Malkawi Z, Griffiths MJ, Scully C. Pulse oximetry during minor oral surgery with and without intravenous sedation. Oral Surg Oral Med Oral Pathol 1992;74(5):537–543.
5. Glassman P, Garrison R. A suggested curriculum for teaching conscious sedation in advanced general practice programs: GPR and AEGD. American Association of Hospital Dentists. Spec Care Dentist 1993;13(1):27–34.
6. Proposed recommended practices. Monitoring the patient receiving I.V. conscious sedation. AORN J 1992;56(2):316–324.
7. Alfonzo-Echeverri EC, Berg JH, Wild TW, Glass NL. Oral ketamine for pediatric outpatient dental surgery sedation. Pediatr Dent 1993; 15(3):182–185.
8. Louon A, Lithander J, Reddy VG, Gupta A. Sedation with nasal ketamine and midazolam for cryotherapy in retinopathy of prematurity. Br J Ophthalmol 1993;77(8):529–530.
9. Coté CJ. Sedation for the pediatric patient: a review. Pediatr Clin North Am 1994;41(1):31–58.
10. Creedon RL. Guidelines for the elective use of conscious sedation, deep sedation, and general anesthesia in pediatric patients. Anesth Prog 1986;33(4):189–190.
11. Nelson MD. Guidelines for the monitoring and care of children during and after sedation for imaging studies. AJR Am J Roentgenol 1993;160(3):581–582.
12. American Academy of Pediatrics, Committee on Drugs. Guidelines for monitoring and management of pediatric patients during and after sedation for diagnostic and therapeutic procedures. Pediatrics 1992; 89(6 Pt 1):1110–1115.
13. Guidelines for the elective use of conscious sedation, deep sedation, and general anesthesia in pediatric patients. ASDC J Dent Child 1986;53(1):21–22.
14. Patterson KW, Casey PB, Murray JP, O'Boyle CA, Cunningham AJ. Propofol sedation for outpatient upper gastrointesinal endoscopy: comparison with midazolam. Br J Anaesth 1991;67(1):108–111.
15. Simon IB, Lewis RJ, Satava RM. A safe method for sedating and monitoring patients for upper and lower gastrointestinal endoscopy. Am Surg 1991;57(4):219–221.
16. Bell GD, McCloy RF, Charlton JE, Campbell D, Dent NA, Gear MW, et al. Recommendations for standards of sedation and patient monitoring during gastrointestinal endoscopy. Gut 1991;32(7):823–827.
17. Harrison BW. Guidelines for care during bronchoscopy. British Thoracic Society. Thorax 1993;48(5):584.
18. Brimacombe J, Berry A. Guidelines for care during bronchoscopy (letter). Thorax 1994;49(5):528.
19. Durbin CG Jr. Sedation for the agitated, critically ill patient without an artificial airway. Crit Care Clin 1995;11(4):913–936.
20. Practice guidelines for sedation and analgesia by non-anesthesiologists. A report by the American Society of Anesthesiologists Task Force on Sedation and Analgesia by Non-Anesthesiologists. Anesthesiology 1996;84(2):459–471.

Correspondence: Charles G Durbin Jr MD, Department of Anesthesiology, University of Virginia Health Science Center, PO Box 10010, Charlottesville Virginia 22908. E-mail: cgd8v@virginia.edu.

Comparison of Conventional Heated Humidification with a New Active Hygroscopic Heat and Moisture Exchanger in Mechanically Ventilated Patients

Richard D Branson RRT, Robert S Campbell RRT, Jay A Johannigman MD,
Michael Ottaway EMT-P, Kenneth Davis Jr MD, Fred A Luchette MD, and Scott Frame MD

BACKGROUND: Humidification has traditionally been supplied by heated humidifiers and, more recently, passive humidifiers. We compared a new active heat and moisture exchanger (AHME) to traditional heated humidification (HH) and heated humidification with a heated wire circuit (HH+HWC). DEVICE STUDIED: The AHME (Humid Heat, Louis Gibeck AB, Sweden) uses a hygroscopic heat and moisture exchanger (HHME) combined with a heat and water source. The HHME is modified to include a cone-shaped housing between the media and the patient. A control unit pumps water to a wick inside the housing, based on the set value for the patient's minute volume. The cone-shaped HHME is inserted into an exterior heater, which only heats the area around the wick. The system is designed to provide an air temperature of 37° C. A temperature probe monitors airway temperature. METHODS: Following informed consent from the next of kin, 30 patients requiring mechanical ventilation for > 72 hours were studied. Patients were ventilated using HH, HH+HWC, and AHME in random order for 24 hours each. All devices were set to deliver gas at 37° C at the proximal airway. Daily measurements were made of minimum and maximum body temperatures, number of suctioning procedures, volume and quality of secretions, volume of instilled saline, number of times condensate was drained from the circuit, water usage, and circuit disconnections. Resistance of the HHME portion of the AHME was measured before and after use. Measurements of relative humidity and temperature at the endotracheal tube were made daily. RESULTS: There were no differences related to the indices of adequate humidification (secretion volume and consistency, number of suctioning attempts, or volume of saline used). All devices provided similar measured airway temperatures and humidity. There were also no differences in the daily minute volume or bias flow for flow-triggering. There were significant differences between devices in water usage per 24 hours (HH = 2039 ± 387 mL, HH+HWC = 766 ± 281 mL, AHME = 135 mL ± 53 mL, $p < 0.001$). The volume of condensate was also significantly less for the HH+HWC and AHME, compared to HH (HH = 930 ± 271 mL, HH+HWC = 12 ± 6, and AHME = 1 ± 3 mL, $p < 0.001$). AHME resistance was relatively unchanged from start to finish (1.7 ± 0.1 cm $H_2O/L/s$ vs 2.3 ± 0.8 cm $H_2O/L/s$). CONCLUSIONS: In this preliminary study, the new AHME provided similar humidification to traditional HH and HH+HWC, but with significantly lower water usage and less condensate than HH or HH+HWC. Disadvantages of the AHME system include added dead space (73 mL) and more weight on the endotracheal tube. However, if a flex tube is used in the HH system, only 54 mL of dead space is added. [Respir Care 1999;44(8):912—917] *Key words: humidification, hygroscopic heat and moisture exchanger, heated wire circuit, mechanical ventilation.*

Introduction

Humidification of inspired gases during mechanical ventilation is a standard of care.[1,2] During the past 4 decades the most common technique for providing humidity during ventilatory support has been heated humidification (HH).

Richard D Branson RRT, Robert S Campbell RRT Jay A Johannigman MD, Michael Ottaway EMT-P, Kenneth Davis Jr MD, Fred A Luchette MD, and Scott Frame MD, are affiliated with the University of Cincinnati College of Medicine, University Hospital, Cincinnati, Ohio.

Mr. Branson presented a version of this paper at the American Association for Respiratory Care Open Forum during the 44th International Respiratory Congress, November 7–10, 1998, in Atlanta, Georgia.

This work was supported by a grant from Louis Gibeck AB, Sweden.

Correspondence: Richard D Branson RRT, University of Cincinnati Medical Center, PO Box 670558, Cincinnati OH 45267-0558. E-mail: Richard.Branson@UC.edu.

During the 1990s, HH with heated wire circuits (HH+HWC) to reduce circuit condensation has become popular. More recently, passive humidifiers or artificial noses have been successfully used with certain patients.[3–5]

Shelley introduced the concept of an active HME in 1991.[6] The active heat and moisture exchanger (AHME) combines the passive operation of a hygroscopic heat and moisture exchanger (HHME) with additional heat and moisture supplied by a heater and water source. This year an AHME (the Humid-Heat by Louis Gibeck AB, Sweden) has been introduced for clinical use. This preliminary study was designed to compare short-term humidification performance by HH and HH+HWC devices and this new AHME device.

Materials and Methods

The study proposal was reviewed by the Institutional Review Board governing human research and approved for the entry of 30 patients. All patients requiring mechanical ventilation for >72 hours in the surgical intensive care unit at the University of Cincinnati were eligible for participation, and written informed consent was obtained from the next of kin prior to study initiation.

Three methods of humidification were studied in random sequence for a period of 24 hours each: (1) heated humidification with a nonheated wire circuit set to deliver gas at 37° C at the proximal airway (MR-730 humidifier, Fisher and Paykel, Auckland, New Zealand); (2) heated humidification with a dual-heated wire circuit set to deliver gas at 37° C at the Y-piece with a temperature of 35° C in the humidification chamber (MR-730 humidifier, Fisher and Paykel, Auckland, New Zealand); and (3) an active heat and moisture exchanger (Humid-Heat, Louis Gibeck AB, Sweden) set to deliver gas at 37° C at the proximal airway. Chambers for the heated humidification systems were the same, and an automatic float-feed, water supply system was used. Extension (flex) tubes between the Y-piece and the artificial airway were used only in patients with tracheostomies.

The following evaluations were performed during each period of ventilatory support. Daily minimum and maximum body temperatures were determined from bedside flow sheets and recorded. Suctioning was performed on an as-needed basis by respiratory care and nursing staff. Suctioning was indicated when secretions were present in the tracheal tube, upon audible evidence of secretions, or when auscultation revealed rhonchi. Suctioning was performed using an open circuit technique and single-use catheters via the sterile technique. The number of suctioning attempts per day was recorded, as well as the volume and quality of secretions, and the volume of instilled normal saline. Secretion volume was measured by collecting secretions in a graduated trap in-line with the suction tubing. Instilled saline was measured by counting the number of unit dose (5 mL) vials used. Secretion quality was based on the scheme of Suzukawa et al,[7] as follows: (1) thin (following the suctioning procedure, the catheter appears clean); (2) moderate (following the suctioning procedure, secretions adhere to the side of the catheter but are easily removed by aspirating saline through the catheter), or (3) thick (following the suctioning procedure, secretions adhere to the catheter and are not removed by aspirating saline through the catheter).

We also recorded the number of circuit disconnections per day. A circuit disconnection was defined as any interruption of the circuit, including suctioning attempts, emptying condensate, changing the circuit or circuit components, removal of the AHME for bronchodilator treatments, and any other circumstance where the circuit was violated. Water usage was determined by weighing the water bags on an analytical balance prior to and after 24 hours of use.

Temperature and humidity were measured daily using a rapid response thermistor and capacitance hygrometer. The hygrometer (Gibeck Humidity Sensor System, Louis Gibeck AB, Sweden) is a capacitance device with an accuracy of ± 4% relative humidity and ± 1% temperature. The 90% response time for relative humidity is 1.4 seconds, and the 90% response time for temperature is less than 150 milliseconds. The sampling rate of the device is 21 Hz. Prior to use, the hygrometer was calibrated using saturated solutions of sodium chloride and lithium chloride. Signals from the hygrometer were imported to a personal computer via an analog-to-digital converter. Real time measurements of temperature and humidity were graphically displayed and the inspired humidity measured as the mean value from the beginning of inspiration to the end of inspiration. Absolute humidity was calculated from relative humidity and temperature according to the following formula:

$$AH = (3.939 + 0.5019T + 0.00004615T^2 + 0.0004188T^3) \times RH/100$$

where AH is absolute humidity, T is temperature, and RH is relative humidity.

Resistance of the HHME portion of the AHME was measured in the laboratory by directing flow from a rotameter at 1.0 L/s through the device and measuring the pressure drop with a differential pressure transducer. This test was made immediately after opening the device packaging, and again after 24 hours of use. After use, the HHME portion of the device was placed in a sealed plastic bag to prevent evaporation of moisture from the device, and transported to the laboratory for testing.

Statistical analysis was performed using analysis of variance to determine differences between devices. A $p < 0.05$ was considered statistically significant.

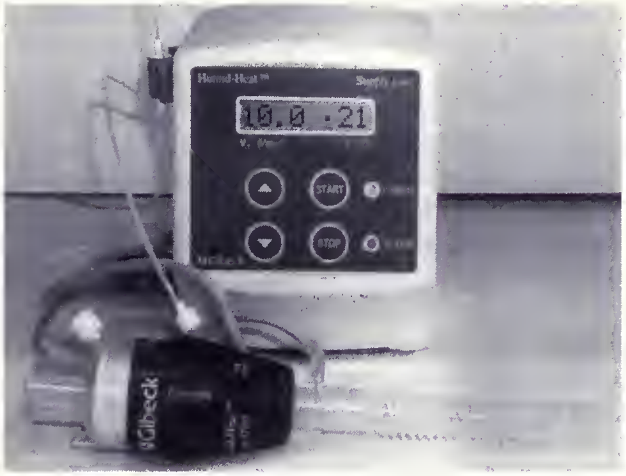


Fig. 1. Active heat and moisture exchanger. Control unit controls both temperature and water supply. Hygroscopic heat and moisture exchanger (forefront of picture), surrounded by the heating unit, is placed between the ventilator circuit and the artificial airway.

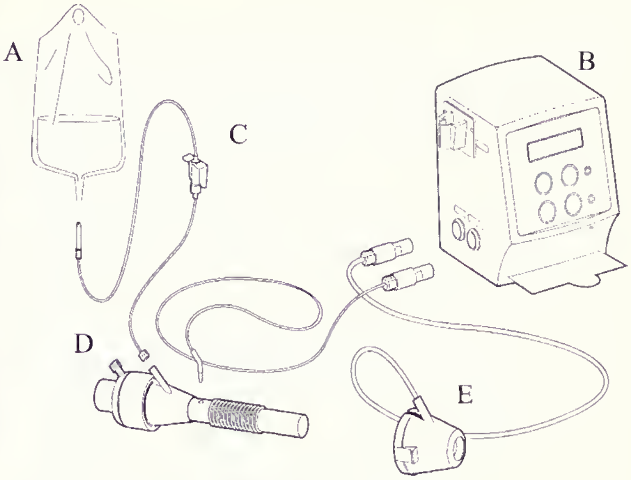


Fig. 2. Components of the active heat and moisture exchanger: (A) Water reservoir bag. (B) Control unit. (C) Feed line from water reservoir bag to hygroscopic heat and moisture exchanger (HHME). (D) HHME modified with a cone-shaped portion lined with a wick for insertion into a heat source. The two lines leading to the HHME are: one from the water source, and one to the temperature probe. (E) Heating unit for the cone-shaped portion.

Device Description

The AHME combines a hygroscopic heat and moisture exchanger with a heated humidifier (Fig. 1 and 2). The components consist of the HHME, a temperature and water supply control unit, a water source and feed line, a heat source, and a temperature probe. The HHME is modified such that connected to the hygroscopically-treated, corrugated paper media is a cone-shaped portion that contains a wick lining the circumference of the internal surface. The control unit pumps water onto the wick. The cone-shaped portion of the HHME is inserted into the heat source. During inspiration, gas travels from the ventilator source through the paper media in the cone-shaped portion, in which additional heat and moisture is added to the inspired gas from the wick surface, and then over the temperature probe. During expiration, any additional heat and moisture added by the wick surface "loads" the HHME. The temperature setting is nonadjustable at 37° C. The control unit adjusts the heating unit and pumps water to the wick surface based on minute ventilation, which is entered by the clinician. If minute ventilation changes and temperature increases or decreases, alarms sound, necessitating a change in the set minute ventilation. If the electronic portion of the device fails or the clinician fails to resupply the water source, the device operates as an HHME, providing approximately 28–31 mg H_2O/L of moisture. Dead space in the device is 73 mL, including a 19 mL flex tube, and resistance is 1.7 cm H_2O/L/s.

Results

Comparisons of the indices of adequate humidification showed no differences between the devices. Secretion volume and consistency, number of suctioning attempts, and volume of saline used were all similar with each humidification device (Table 1). All devices provided similar measured airway temperatures and humidity. There were also no differences in the daily minute volume or bias flow for flow-triggering. Daily minute volume for HH, HH+HWC, and AHME were 11.6 ± 3.3, 11.9 ± 3.4, and 11.8 ± 2.7 L/min, respectively. Bias flow for flow-triggering was also similar: 5.8 ± 2.5, 5.4 ± 2.6, and 5.9 ± 2.3 L/min, respectively.

There were significant differences between the devices with regard to water use per 24 hours (HH = 2039 ± 387 mL, HH+HWC = 766 ± 281 mL, AHME = 135 ± 53 mL, $p < 0.001$ for HH vs HH+HWC and for HH+HWC vs AHME, and $p < 0.0001$ for HH vs AHME). The volume of condensate was also significantly less for HH+HWC and AHME than for HH (HH = 930 ± 271 mL, HH+HWC = 12 ± 6 mL, and AHME = 1 ± 3 mL, $p < 0.001$ for HH vs HH+HWC and AHME). Condensate collected from the AHME system was drained from the flex tube between the device and the patient. There was no condensate found in the ventilator circuit between the AHME and the ventilator. AHME resistance was relatively unchanged from start to finish (1.7 ± 0.1 cm H_2O/L/s vs 2.3 ± 0.8 cm H_2O/L/s).

Measurements of airway temperature and humidity were similar for each device (see Table 1). There were no complications of humidifier use in any of the patients studied. Alarm conditions for each type of device were similar based on written reports by the respiratory therapy staff. The most common alarms for HH and HH+HWC were

Table 1. Comparison of Measured Variables Using the Three Humidification Devices Described in the Study

	Heated Humidifier	Heated Humidifier with Heated Wire Circuit	Active Heat and Moisture Exchanger
Body temperature max/min (°C)	38.6 ± 1.1/36.2 ± 0.5	38.1 ± 1.3/36.5 ± 1.0	38.2 ± 0.8/36.6 ± 0.9
Volume of secretions (mL)	30 ± 22	30 ± 20	31 ± 21
Number of suctioning attempts	6.3 ± 2.1	6.2 ± 2.0	6.3 ± 2.2
Number of saline instillations	3.3 ± 1.6	3.0 ± 1.1	3.9 ± 1.4
Secretion viscosity	1.4 ± 0.3	1.3 ± 0.5	1.3 ± 0.6
Water usage (mL/24 h)	2039 ± 387*†	766 ± 281	135 ± 53‡
Condensate (mL/24 h)	930 ± 271*†	12 ± 16	1 ± 3‡
Airway Temperature (°C)	36.3 ± 1.2	37.1 ± 1.0	36.4 ± 1.7
Relative Humidity (%)	97 ± 4	93 ± 5	98 ± 3
Absolute Humidity (mg H_2O/L)	40.6 ± 3.1	38.2 ± 4.2	41 ± 3.0

---

*$p < 0.001$ HH vs HH + HWC.

† $p < 0.001$ HH vs AHME.

‡ $p < 0.01$ HH + HWC vs AHME.

related to empty humidification chambers due to kinks in the feed hose or empty feed sets. The most common alarms for the AHME occurred when minute volume changes resulted in a change in delivered temperature.

Discussion

Our results indicate that the AHME can provide heat and humidification equivalent to conventional heated humidification, but with lower water consumption and less condensate. During use, the HHME provides approximately 28–31 mg H_2O/L of moisture. The desired humidifier output is 44 mg H_2O/L (37° C at 100% relative humidity), so the heated portion of the device only needs to contribute 13–16 mg H_2O/L to the inspired gas.

In contrast, the heated systems have to supply the entire 44 mg H_2O/L. With an HH and nonheated wire circuit, chamber temperature often must be kept at 50° C in order to delivery 37° C gas at the airway. The resulting drop in temperature along the length of the circuit creates a large volume of condensate. The HH+HWC essentially eliminates condensate by increasing temperature along the length of the tubing, but this decreases the relative humidity. At reasonable temperature differences (< 3° C) this reduction in relative humidity appears to be clinically unimportant. However, at extremes of temperature differences (3–6° C), the reduction in relative humidity can cause drying of airway secretions, and airway obstruction.[8–10]

Another reason for the lower water usage is the position of the AHME relative to the HH. In our intensive care unit, flow-triggering has become ubiquitous. In fact, several new ventilators produce a baseline continuous flow, even in the absence of flow-triggering. A conventional humidifier warms and humidifies baseline flow, despite the fact that some of the gas passes directly through the circuit without reaching the patient. The position of the AHME, between the endotracheal tube and ventilator circuit, removes it from the path of the baseline flow. In this study, the mean baseline flow was 5 L/min. If this gas is humidified to 44 mg H_2O/L at the humidifier outlet, then an additional 316,800 mg of H_2O would be added (5 L/min × 1440 min/d × 44 mg H_2O/L). This alone equates to an additional water consumption of over 300 mL per day. When a conventional HH with a nonheated wire circuit is used, water consumption increases dramatically. However, because the baseline flow only occurs during the expiratory phase, the actual additional water consumption depends on respiratory frequency, inspiratory time, and inspiration-expiration ratio.

Limitations of the AHME include: nonadjustablity of the proximal airway temperature, increased dead space, and the added weight of the device. These will be considered below.

We and others have argued that the appropriate delivered temperature and relative humidity for mechanically ventilated patients reestablishes normal physiologic conditions.[11,12] We believe this temperature to be 32–34° at 95–100% relative humidity. The AHME studied herein provides humidity in excess of these target values, but we recognize that patients with lung disease, fluid restriction, and chronic sputum production may need higher levels of humidification for restoration of normal conditions. In this study we did not observe any adverse effects from the 37° C temperature, and others have suggested that a temperature of 37–40° C is appropriate.[13]

Our approach to humidification in the intensive care unit utilizes both HH and HHME—the selection is based on the patient's temperature, secretion quality, presence of blood in the sputum, history of chronic lung disease, and duration of ventilation.[3,12–14] Dreyfuss et al studied humid-

Table 2. Advantages and Disadvantages of Humidification Devices

	Advantages	Disadvantages
Heated Humidification	• Wide range of temperatures • Universal application • Reliable • Temperature monitoring • Alarms	• Excess condensate • Cost • Maintenance • Possibility of over-heating • Remote possibility of electric burns/shock • Lack of portability
Heated Humidification with with heated wire circuit	• Wide range of temperatures • Universal application • Reliable • Temperature monitoring • Alarms • Elimination of condensate	• Potential for reduced relative humidity and airway obstruction • Cost (if used < 48 hrs) • Lack of portability • Complexity
Passive Humidifier	• Passive operation • Portable • Light weight • Simple • Cost	• Net water loss from respiratory tract • Not suitable for all patients • Deadspace • Resistance • Potential for occlusion
Active Heat and Moisture Exchanger	• Universal application • Low water consumption • Elimination of condensate • Temperature monitoring • Alarms • Continued passive operation if electricity or water source is lost • Elimination of water traps/heated wires	• Deadspace • Weight • Potential for occlusion • Limited temperature range • Heat source near patient

ification in the medical intensive care unit and reported using HHME in all patients for an extended period.[4] At the other extreme of the spectrum, Lawrence reports using only HH+HWC and airway temperature of 40° C.[13] Each of these investigators reported safe, effective application of these humidification techniques. Optimum temperature and humidity remain elusive concepts because of variability among patients and diseases, but, taken together, these results suggest that the AHME may play a role in bridging the gap between those who currently use only active humidification and those who use only passive humidification.

The dead space of a heat and moisture exchanger can negatively impact ventilation by increasing the ratio of dead space to tidal volume,[15–17] and, in spontaneously breathing patients, the work of breathing may increase.[16] This is one of the disadvantages of traditional HHME use and will also impact the use of the AHME in select patients. The total dead space of the AHME is 73 mL. The flex tube between the device and the patient accounts for 19 mL of this volume. If a flex tube is already in place for a traditional heated humidification system, the AHME adds only 54 mL of dead space. Added dead space may have adverse clinical effects during weaning and in patients with marginal respiratory muscle function, but the use of pressure support may mitigate this problem with both HHME and the AHME.[17]

The total weight of the HHME and heating unit is 79 g, approximately twice that of a typical HHME. The HHME portion weighs 36 g and the heating unit weighs 27 g. Although a few large dead space HHMEs (100 mL) weigh up to 50 g, the weight of the AHME must be considered. The heating unit has an attachment that connects to a typical ventilator circuit support arm. During the study we used the support arm as well as simply rested the device on a towel near the patient's chest. During the study, no airway complications were noted during AHME use, but the added weight of the AHME should be considered when positioning the device. The potential for inadvertent extubation created by the added weight was not considered in this initial study. This may be a matter of concern, but we believe it can be dealt with effectively by careful clinicians.

The external temperature of the heating unit was measured at 38° C. This temperature is not typically associated with skin burns. However, patients with hypotension and poor skin perfusion could be at risk for thermal injury. The AHME heating unit should not be allowed to rest directly on the patient's skin. In our study, we did not experience any problems with the external temperature of the AHME.

Table 2 lists advantages and disadvantages of humidification devices. The choice of humidification device is dictated by patient requirements, device performance, and cost. Introduction of the AHME allows clinicians a third humidification choice. We found some advantages to the AHME compared to HH and HH+HWC, but the cost of the AHME is not yet known, so cost comparisons are not yet possible.

Conclusions

This preliminary study compared a new humidification device, the AHME, to traditional HH and HH+HWC. Measurements of delivered temperature and humidity, as well as indices of humidification adequacy found no difference between the devices. However, the AHME was associated with reduced water consumption and reduced condensate formation, which might offer some cost savings. The added dead space of the AHME poses a concern in certain patients, as it does with traditional HHMEs. The AHME provides a new humidification method that bridges the gap between active and passive humidification.

ACKNOWLEDGMENTS

The authors are grateful for thoughtful review of the manuscript by Hans Lambert and Lena Tibell, Louis Gibeck AB, Sweden.

REFERENCES

1. American Association for Respiratory Care. Consensus statement on the essentials of mechanical ventilators—1992. Respir Care 1992; 37(9):1000–1008.
2. AARC Clinical Practice Guideline. Humidification during mechanical ventilation. Respir Care 1992;37(8):887–890.
3. Branson RD, Davis K Jr, Campbell RS, Johnson DJ, Porembka DT. Humidification in the intensive care unit. Prospective study of a new protocol utilizing heated humidification and a hygroscopic condenser humidifier. Chest 1993;104(6):1800–1805.
4. Dreyfuss D. Djedaini K, Gros I, Mier L, Le Bourdelles G, Cohen Y, et al. Mechanical ventilation with heated humidifiers or heat and moisture exchangers: effects on patient colonization and incidence of nosocomial pneumonia. Am J Respir Crit Care Med 1995;151(4): 986–992.
5. Djedaini K, Billiard M, Mier L, Le Bourdelles G, Brun P, Markowicz P, et al. Changing heat and moisture exchangers every 48 hours rather than 24 hours does not affect their efficacy and the incidence of nosocomial pneumonia. Am J Respir Crit Care Med 1995;152(5 Pt 1):1562–1569.
6. Shelly MP. Humidification in the future. Probl Respir Care 1991; 4(4):487–491.
7. Suzukawa M, Usada Y, Numata K. The effects on sputum characteristics of combining an unheated humidifier with a heat-moisture exchanging filter. Respir Care 1989;34(11):976–984.
8. Miyao H, Miyasaka K, Hirokawa T, Kawazoe T. Consideration of the International Standard for airway humidification using simulated secretions in an artificial airway. Respir Care 1996;41(1):43–49.
9. Miyao H, Hirokawa T, Miyasaka K, Kawazoe T. Relative humidity, not absolute humidity, is of great importance when using a humidifier with a heating wire. Crit Care Med 1992;20(5):674–679.
10. Gradon L. Relative humidity with heating wire (letter). Crit Care Med 1993;21:1613–1615.
11. Chatburn RL, Primiano FP Jr. A rational basis for humidity therapy (editorial). Respir Care 1987;32(4):249–254.
12. Branson RD, Campbell RS. Humidification in the intensive care unit. Respir Care Clin N Am 1998;4(2):305–320.
13. Lawrence JC. Humidification practices in the adult intensive care unit, Prince of Wales Hospital (review). Respir Care Clin N Am 1998;4(2):301–304.
14. Branson RD, Davis K Jr, Brown R, Rashkin M. Comparison of three humidification techniques during mechanical ventilation: patient selection, cost, and infection considerations. Respir Care 1996;41(9): 809–816.
15. Le Bourdelles G, Mier L, Fiquet B, Djedaini K, Saumon G, Coste F, Dreyfuss D. Comparison of the effects of heat and moisture exchangers and heated humidifiers on ventilation and gas exchange during weaning trials from mechanical ventilation. Chest 1996;110(5):1294–1298.
16. Iotti GA, Olivei MC, Palo A, Galbusera C, Veronesi R, Comelli A, et al. Unfavorable mechanical effects of heat and moisture exchangers in ventilated patients. Intensive Care Med 1997;23(4):399–405.
17. Pelosi P, Solca M, Ravagnan I, Tubiolo D, Ferrario L, Gattinoni L. Effects of heat and moisture exchangers on minute ventilation, ventilatory drive, and work of breathing during pressure support ventilation in acute respiratory failure. Crit Care Med 1996;24(7):1184–1188.

Effects of Tracheal Gas Insufflation and Tracheal Gas Exsufflation on Intrinsic Positive End-Expiratory Pressure and Carbon Dioxide Elimination

Toshimichi Takahashi MD, Guillermo Bugedo MD, Alexander B Adams RRT MPH,
Peter L Bliss BME, and John J Marini MD

BACKGROUND: Tracheal gas insufflation (TGI) reduces arterial carbon dioxide tension (P_{aCO_2}) when used in conjunction with mechanical ventilation, but antegrade TGI flow increases intrinsic positive end-expiratory pressure (auto-PEEP) or end-expiratory lung volume (EELV). Tracheal gas exsufflation (TGE) might reduce TGI-elevated EELV while retaining the CO_2 elimination effects of TGI. METHODS: An apparatus was designed to control TGI and/or TGE during selected portions of expiration. The first phase of the study examined the general effects of expiratory TGI and TGE on EELV and CO_2 elimination in a mechanical test lung. In the second phase, effects on normal physiology were tested in uninjured dogs. Changes in EELV were measured by respiratory inductance plethysmography, and P_{aCO_2} was continuously monitored. TGI and TGE were applied separately and in combination during all or half of the expiratory phase. TGI/TGE combinations found to be most effective at CO_2 elimination in the test lung model were applied in the animal tests. RESULTS: TGI raised EELV and TGE lowered EELV in both models, and exerted their greatest effects on EELV in the latter half of expiration. In the animal model, the TGI/TGE interventions tested reduced P_{aCO_2} by 16–32% ($p < 0.01$) compared to baseline P_{aCO_2}, similar to or greater than reported clinical studies. CONCLUSIONS: Combining TGI and TGE allowed precise control of EELV in both models. Second-half TGI provided the most effective CO_2 elimination. [Respir Care 1999;44(8):918–924] *Key words: tracheal gas exsufflation, tracheal gas insufflation, auto-PEEP, CO_2 elimination, lung model.*

Background and Objectives

In prior studies,[1–7] tracheal gas insufflation (TGI) has been shown to reduce arterial carbon dioxide tension (P_{aCO_2}) when used in conjunction with mechanical ventilation. TGI's reduction of P_{aCO_2} varies depending on the pathology of the lung, the volume of fresh gas injected per breath cycle, and the initial degree of hypercapnia.[1,8–9] TGI directed toward the alveoli (antegrade flow) has two CO_2 elimination effects: distal washout (beyond the catheter tip) and proximal (before the catheter tip) washout. As a gas source that opposes the direction of expiratory flow, gas injected toward the carina by TGI has the potential to increase end-expiratory lung volume (EELV) (as reflected by intrinsic positive end-expiratory pressure [auto-PEEP]).[1,2,10–12] While antegrade TGI flow increases EELV, retrograde TGI flow (via reverse flow catheters) avoids increasing EELV but might not obtain the distal CO_2 elimination effect. The use of tracheal gas exsufflation (TGE) in conjunction with TGI might allow reduction and control of EELV while preserving the distal effect.

In the late 1940s and early 1950s, TGE was first used in conjunction with positive pressure ventilation[13–15] and was found to counter the adverse effects of positive pressure ventilation on cardiac output.[13] TGE augmented venous return and reduced lung volume, but its use was aban-

---

Toshimichi Takahashi MD was affiliated with the section of Pulmonary/Critical Care, Regions Hospital and University of Minnesota, St. Paul, Minnesota at the time this study, and is now affiliated with the Department of Anesthesiology, Nagoya University School of Medicine, Nagoya, Japan. Guillermo Bugedo MD, Alexander B Adams RRT MPH, Peter L Bliss BME, and John J Marini MD are affiliated with the Section of Pulmonary/Critical Care, Regions Hospital and University of Minnesota, St Paul, Minnesota.

Support: NIH SCOR 5P50-HL50152–03

Correspondence: Alexander B Adams RRT MPH, Pulmonary Research, Regions Hospital, 640 Jackson Street, St Paul MN 55101. E-mail: alex.b.adams@healthpartners.com.

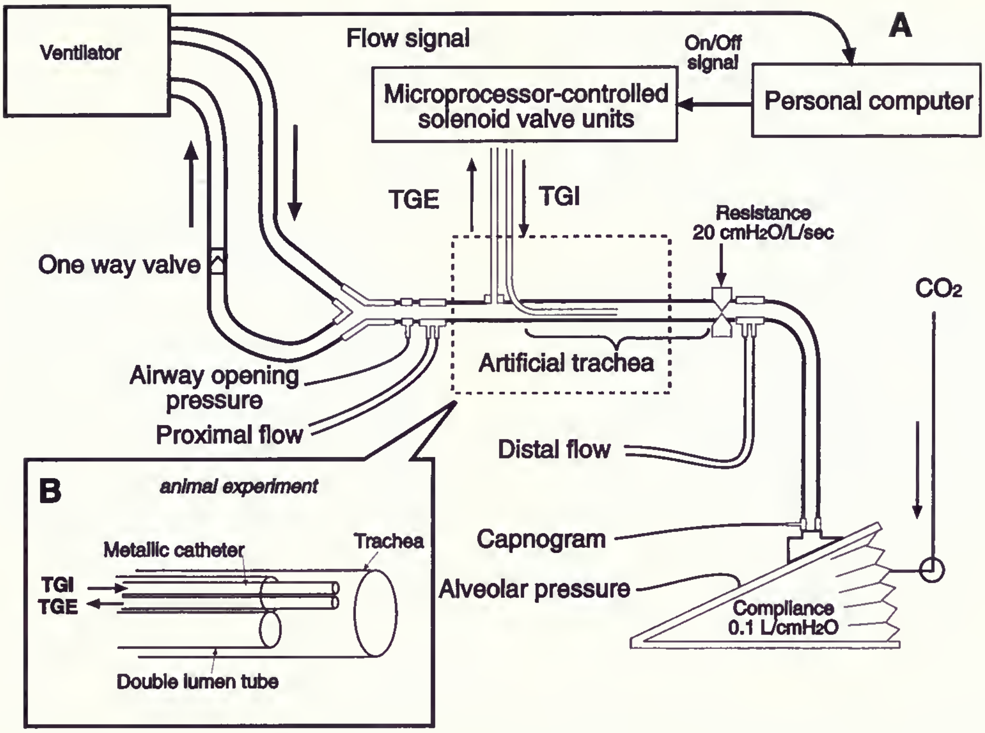


Fig. 1. Schematics of the tracheal gas insufflation/tracheal gas exsufflation (TGI/TGE) experimental settings. A: Pneumatic circuitry for lung model experiments. B: Close-up of the endotracheal tube and TGI/TGE catheters for the animal experiments.

doned because of its potential to accentuate airway closure.[16] Not previously investigated is the possibility that TGE might also improve CO_2 elimination if applied selectively during portions of expiration. In concept, TGE might fill the central airways with CO_2-laden alveolar gas earlier in expiration for TGI-accelerated removal. Recent advances in the technology of solenoid valves and microprocessing allow sophisticated control of airway pressure, potentially facilitating timed application of TGI and TGE in the clinical setting.[17–18]

We investigated the effects of expiratory TGI and TGE used (independently and together) on auto-PEEP and CO_2 elimination. General effects were initially tested in a mechanical lung model, followed by testing of short-term physiologic effects in uninjured dogs.

Materials and Methods

Apparatus

We designed a double-solenoid, microprocessor-controlled device to precisely gate expiratory TGI and TGE, guided by flow signals from the ventilator circuit (Fig. 1). A standard flow meter and portable suction pump provided the flow, and either TGI, TGE, or TGI and TGE together were delivered, either throughout expiration, during the first half of expiration, or during the second half of expiration. The process was controlled by a personal computer (Gateway 2000, 486/33 MHz, North Sioux City, South Dakota). Prior to use in these protocols, the device was thoroughly tested in a bench setting and calibrated to gate constant positive (TGI) and/or negative (TGE) flows.

Methods

For the lung model study, a system was constructed (see Figure 1A) using a single-compartment lung model (TTL Model 1600, Michigan Instruments Inc, Grand Rapids, Michigan), an artificial trachea, and a mechanical ventilator (7200ae, Mallinckrodt, Pleasanton, California). The lung compliance was set at 0.1 L/cm H_2O, and a linear resister of 20 cm H_2O/L/s was positioned in the common portion of a standard ventilator circuit. The ventilator settings were: tidal volume (V_T) = 600 mL, breaths per minute (f) = 18/min, constant inspiratory flow = 40 L/min. The outcome variables measured were EELV and CO_2 elimination rate.

We evaluated, in random sequence, a grid of 16 interventions that tested TGI or TGE alone, or TGI and TGE together, for the entirety of the expiration, for the first half of expiration, for the second half of expiration, and for none of the expiration. Both TGI (positive) and TGE (negative) flows were fixed at 9 L/min (constant flow). The auto-PEEP resulting from each intervention was recorded with a chart recorder (95000, Astro-Med, West Warwick, Rhode Island) and stored on digital audio tape (RD-111T, TEAC, Tokyo, Japan) for later analysis. External positive end-expiratory pressure (external PEEP) was set at 5 cm H_2O as a baseline from which changes in EELV and pressure caused by the intervention were determined in triplicate and averaged for data analysis. EELV was directly reflected in the test lung by the end-expiratory bellows pressure (auto-PEEP).

The second phase of the lung model study involved infusing CO_2 (1–2 L/min) into the cycling test lung system, without TGI or TGE, until a steady state end-tidal carbon dioxide pressure ($P_{ET}CO_2$) of 70 mm Hg (9.3 kPa) was detected by the capnograph (Novametrix 2000; Wallingford, Connecticut). The baseline CO_2 elimination rate was determined by measuring $P_{ET}CO_2$ at 10-second intervals after the CO_2 infusion to the test lung was stopped. The same procedure was then followed for each TGI/TGE intervention. The $P_{ET}CO_2$ measurements were plotted against time as an index for CO_2 elimination. Measurements were repeated twice and the averaged values were used for analysis.

Since TGI should raise EELV and TGE should lower EELV, these interventions will expand or reduce the CO_2 reservoir existing at the onset of the washout period. This change in EELV, as a separate factor, alters the apparent CO_2 elimination rate during tidal ventilation. To determine CO_2 elimination rates independent of this effect on EELV, we measured EELV change by each of the TGI/TGE interventions and matched baseline EELV for each intervention by adjusting external PEEP (about the baseline external PEEP of 5 cm H_2O).

The animal model portion of the study involved the instrumentation and monitoring of 6 normal, supine mongrel dogs (24.6 kg ± 2.0). The dogs were intravenously anesthetized with pentobarbital sodium (30 mg/kg), paralyzed with pancuronium bromide (0.1 mg/kg), and maintained with continuous infusions of 3–5 mg/kg/h and 0.1 mg/kg/h, respectively, until the end of the experiment. Femoral arterial and venous lines were placed, and a pulmonary artery catheter was placed via the internal jugular vein. A tracheotomy was performed to assure stable access to the airway, using a double-lumen endotracheal tube (41 French left, Sheridan, St Louis, Missouri). One lumen of the endotracheal tube was used for conventional ventilation and the other served as a secure conduit for the TGI and TGE catheters (Figure 1B). The animals were mechanically ventilated (7200ae, Mallinckrodt, Pleasanton, California) in the volume-controlled mode: fraction of inspired oxygen (F_{IO_2}) = 0.4, V_T = 10 mL/Kg, constant flow = 10 L/min, external PEEP = 5 cm H_2O, f = 10/min. Heated pneumotachographs were placed in the inspiratory and expiratory limbs of the circuit. The dogs were prepared and monitored according to a protocol approved by the animal care and use committee of our institution. Arterial blood gas concentrations were continuously sensed and recorded by an indwelling fiberoptic system (Paratrend 7, Diametrics, St Paul, Minnesota).

Under bronchoscopic guidance, 2 side-by-side copper catheters (outside diameter = 3.2 mm, inside diameter = 1.6 mm) were positioned with tips at 1 centimeter above the carina. The position was secured for the duration of the study. The interventions of the test lung study were then applied by the double-solenoid, gated microprocessor-controlled system. TGI and TGE flows were square wave (constant flow) at ± 9 L/min. For the TGI catheter flow, air and O_2 were blended to achieve the same F_{IO_2} (0.40) as the ventilator. Changes in lung volume were indexed by the change in EELV measured by the sum curve of a calibrated (least squares method) respiratory inductive plethysmograph. These changes were recorded with a chart recorder and stored on digital audio tape for later analysis.

The results from the lung model study guided the selection of interventions tested in the animal study. The effect of TGI/TGE on EELV was determined for all interventions in the grid. The effect of the interventions on CO_2 elimination was confined to combinations found to produce the greatest effects in the lung model study while avoiding late TGE, because of TGE's potential for airway closure. These interventions were (1) TGE in the first half of the expiratory phase and TGI in the second half of the expiratory phase (1st TGE/2nd TGI); (2) 2nd TGI only; and (3) full TGI/full TGE. CO_2 elimination was evaluated for these selected interventions by calculating the percent change from baseline P_{aCO_2}. Percent change was determined to account for variability in baseline P_{aCO_2} and to compare the intervention to clinical studies. After an intervention, a 40-minute stabilization period was allowed before P_{aCO_2} was recorded from the continuous arterial blood gas analyzer. The baseline ventilator settings were F_{IO_2} = 0.4, V_T = 10 mL/Kg, inspiratory flow = 10 L/min (constant flow), external PEEP of 5 cm H_2O, and a frequency (6–14/min) to produce a moderate hypercapnia ($P_{aCO_2} \approx 60$ mm Hg [8.0 kPa]). Both TGI and TGE flow settings were reduced to ± 4.5 L/min (constant flow) for this stage to limit intervention-caused lung volume excursions to less than the set V_T. Immediately after the intervention, external PEEP was adjusted to maintain a constant EELV (as measured by respiratory inductive plethysmography) to assure that CO_2 elimination was not affected by a change in EELV (the CO_2 reservoir). Another stage was added to match the increased lung volume excursion of 1st TGE/2nd TGI by ventilating at that increased V_T level without an intervention.

The effect of the selected TGI/TGE interventions on P_{aCO_2} change between groups was analyzed by one-way factorial analysis of variance (ANOVA). Significance was accepted at $p < 0.01$ using Fisher's Test (protected least

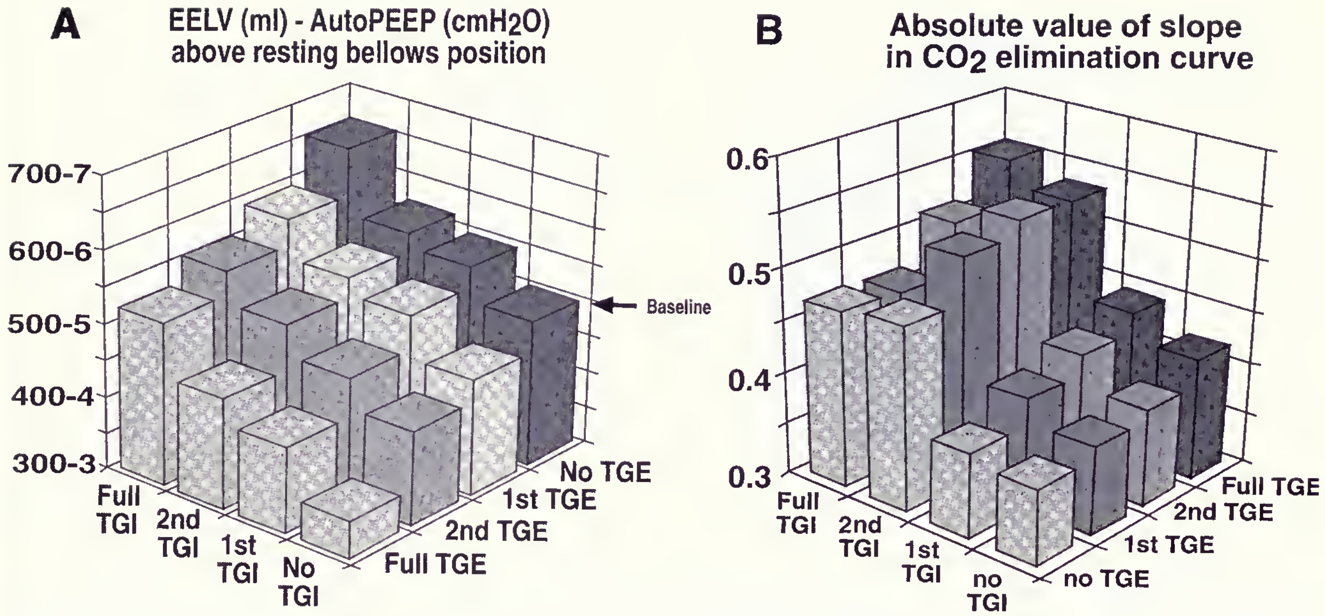


Fig. 2. The effects of tracheal gas insufflation/tracheal gas exsufflation (TGI/TGE) on end-expiratory lung volume (EELV) and CO_2 elimination in the mechanical test lung. A: Changes in EELV by TGI/TGE show their additive/subtractive effects on EELV. AutoPEEP = intrinsic positive end-expiratory pressure. B: An absolute value for a curve fit of the slope of the CO_2 elimination rate was calculated: positive slopes indicate more rapid CO_2 elimination. The values for CO_2 elimination rate slopes are shown for all TGI/TGE combinations.

squares difference) to identify differences in P_{aCO_2} from baseline and between interventions.

Results

Figure 2 shows the effects of tested interventions on EELV and CO_2 elimination from the lung model study. Figure 2A shows, as expected, that TGI consistently increased EELV and TGE consistently decreased EELV. For both, the effect on EELV was consistently greater when applied during the second half of exhalation. Because CO_2 elimination from the lung model occurred exponentially, we plotted CO_2 elimination slopes on a semi-logarithmic scale to curve fit the slope and characterize the curves with one value for intercurve comparisons. Figure 2B shows a CO_2 elimination rate slope histogram for all TGI/TGE combinations tested. TGI had a greater effect on CO_2 elimination during the second half of exhalation, with little effect in the first half. TGE effect on CO_2 elimination was less than the TGI effect, and slightly greater in the second half than in the first half. The greatest effects on CO_2 elimination were seen with full TGI/full TGE. The 2nd TGI/any TGE was the next most effective in CO_2 elimination. The 2nd TGI was third most effective.

Figure 3A shows the effects of each intervention on EELV in the animal model study. The tendencies in the animal study were identical to those of the test lung study. Figure 3B shows the CO_2 elimination effect of the tested interventions. The greatest reduction (–32%) was found with the combination of 1st TGE/2nd TGI ($p < 0.01$). The 2nd TGI (–22%) and full TGI/full TGE (–16%) interventions also caused significant reductions in P_{aCO_2} ($p < 0.01$). Although the set ventilator V_T was maintained constant throughout each study, the combination of 1st TGE/2nd TGI increased lung volume excursion compared to baseline, which might account for its additional CO_2 elimination effect (Fig. 4). The V_T excursion (Fig. 4A) was increased by the intervention (Fig. 4B), effectively resulting in an increased minute ventilation. To compare the intervention 1st TGE/2nd TGI to a ventilator-increased V_T, we increased the set pre-intervention V_T excursion (Fig. 4C), without TGI or TGE applied, to match the intervention V_T excursion. The reduction in P_{aCO_2} of the intervention remained significantly greater than the matched V_T excursion intervention ($p < 0.01$). Throughout the study, we observed no significant changes in arterial oxygen tension (P_{aO_2}) (pre-study P_{aO_2} = 203.3 mm Hg, post-study P_{aO_2} = 210.5 mm Hg), or other monitored variables.

Discussion

Although TGI has the potential to reduce ventilatory requirements (V_T and/or ventilatory pressure), the clinical use of TGI must account for the fact that antegrade TGI can induce or increase EELV, for several reasons, including: momentum transfer between the jet stream and residual gas in the alveoli; increased back pressure to lung emptying; flow across the endotracheal tube and circuit

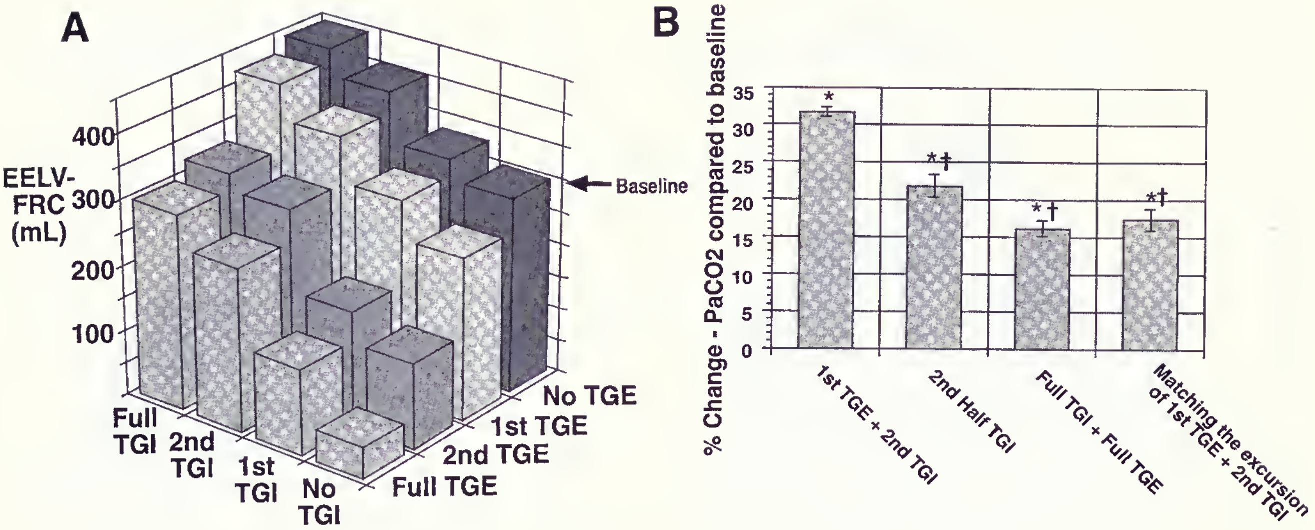


Fig. 3. The effects of tracheal gas insufflation (TGI) and tracheal gas exsufflation (TGE) on CO_2 elimination in the lung model experiment. EELV = end-expiratory lung volume. 1st = first half of expiration. 2nd = second half of expiration. A: Changes in EELV - functional residual capacity (EELV-FRC) by TGI/TGE show similar additive/subtractive effects to the lung model study. B: Percent changes in P_{aCO_2} compared to baseline for the animal experiment. The increases in CO_2 elimination for the 4 conditions tested were significantly greater than baseline. 1st TGE + 2nd TGI had a greater CO_2 elimination rate than the other interventions. Values are mean ± standard deviation. $p < 0.01$ versus baseline. † $p < 0.01$ versus 1st TGE + 2nd TGI.

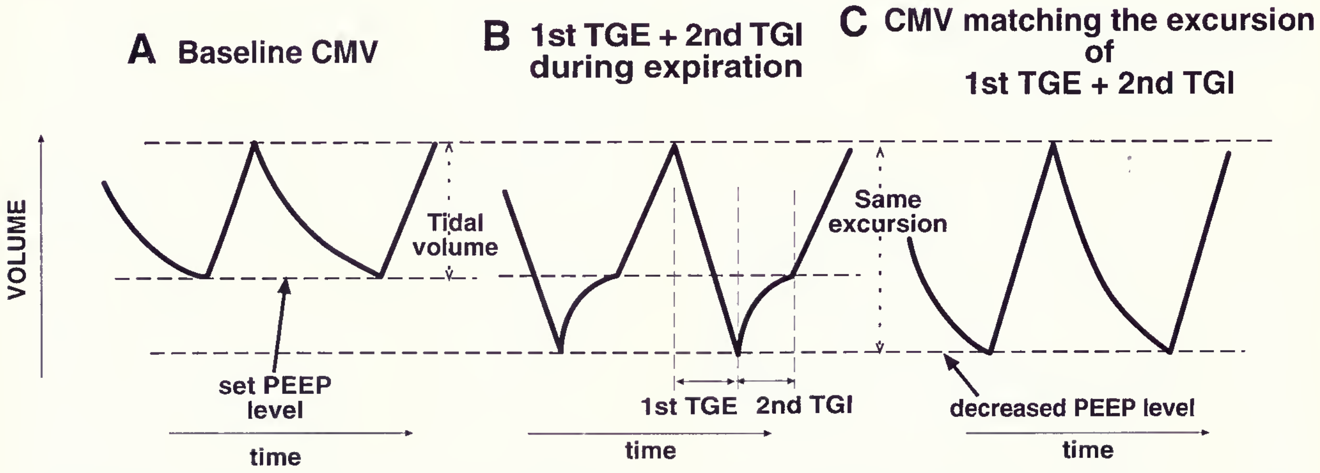


Fig. 4. Schematic representation of changing lung volume for 3 conditions. CMV = constant minute ventilation. 1st = first half of expiration. TGE = tracheal gas exsufflation. 2nd = second half of expiration. TGI = tracheal gas insufflation. PEEP = positive end-expiratory pressure. A: Baseline with conventional constant flow ventilation. B: Applying 1st TGE/2nd TGI displaying increased V_T excursion. C: Conventional constant flow ventilation matching tidal volume excursion of 4B.

elements; and decreased cross-sectional area because of the resistance of the TGI catheter positioned in the airway. Ravenscraft et al[1] reported that in 8 patients EELV increased by an average of 62 mL during 6 L/min antegrade TGI flow. Belghith et al[19] reported that in 8 patients EELV increased by 200 mL during 4 L/min antegrade TGI flow. Imanaka et al[20] also reported increased EELV during antegrade TGI. Several investigators have tested reverse flow catheters designed to avoid increasing EELV, but these designs may reduce the distal CO_2 elimination effect.[21–24]

The present study investigated a means of controlling EELV by specifically using TGE to reduce TGI-augmented EELV. An anticipated result of this study was that the opposing effects of TGI and TGE on EELV allowed precise control of EELV in both settings. In this study the effects of TGI/TGE on EELV were greatest when applied during the second half of exhalation. In both models, expiratory flow from the lung during the first half of exhalation was greater than the set TGI or TGE flows. Thus, during the first half of exhalation the TGI/TGE effect is dominated by expiratory flow from the lungs. On the other hand, the effects of TGI/TGE on EELV are proportionately greater during the second half of exhalation, when flow from the lung is markedly reduced or completed. Therefore, TGI increases EELV and TGE decreases EELV primarily when applied during late exhalation. The small differences in effects on EELV between the 2 models could be due to the nonlinear impedance of the animal model and the different locations of the TGE port between models.

TGI improves the efficiency of CO_2 elimination accomplished by conventional mechanical ventilation, primarily by reducing the anatomic dead space volume distal and proximal to the catheter tip.[25–32] For each intervention in the lung volume study, the slope of the CO_2 elimination curve was steeper than the baseline curve. Thus, in the mechanical model, TGI and TGE both eliminated CO_2 at a greater rate than baseline. In the animal model, P_{aCO_2} was markedly reduced by 1st TGE/2nd TGI (–32%). The CO_2 reduction by the other interventions (–22% and –16%) was similar to or greater than reductions reported in clinical studies.[1,8] Total tidal lung volume excursion increased during 1st TGE/2nd TGI (Fig. 4B) because lung volume was reduced by TGE below EELV in early exhalation and EELV was restored by TGI during late exhalation. This expiratory event added to minute ventilation. The P_{aCO_2} level of the matched excursion level was determined and compared to 1st TGE/2nd TGI. During 1st TGE/2nd TGI the P_{aCO_2} reduction was found to be significantly greater than the matched excursion level (see Fig. 4). Therefore, the effects of 1st TGE/2nd TGI were responsible for increased CO_2 elimination over and above the lung volume excursion increase. Yet, 2nd TGI also exerts a significant P_{aCO_2} reduction effect, and might be as effective as 1st TGE/2nd TGI with a matched volume excursion.

In both models, TGI most effectively eliminated CO_2 when applied in late expiration. As a mechanism to flush the region proximal to the injection site, TGI applied during the *earliest part of exhalation* should be ineffective at CO_2 removal, since it would simply eliminate a segment of fresh gas from the previous inhalation. Similarly, TGI applied selectively during *mid-expiration* would flush alveolar CO_2 that would have been expelled without it. As expected, TGI was most effective during *late expiration*, since it flushes CO_2 from the anatomical dead space that would normally remain to be rebreathed on the next inspiration. This finding supports our earlier work demonstrating the importance of flushing the terminal phase of expiration, where TGI flush volume becomes relevant.[7]

For the animal experiments, 6 hours of study were conducted, with several TGE interventions. In these normal animals, no reduction of P_{aO_2} that might have been caused by airway closure was seen after 6 hours of study involving TGE. In the animals, any TGE-induced airway closure would tend to occur near the end of exhalation, and we did not specifically test this possibility. Whether the result would have been different in the setting of unstable airways is not known. Moreover, we studied only 2 levels of TGI and TGE applied to a fixed pattern of ventilation. It is not possible to determine from the data currently available whether qualitative differences would have emerged under other ventilatory patterns and TGI/TGE flows.

In summary, we observed that combined TGI and TGE can improve CO_2 elimination while controlling EELV. We confirmed that TGI was most effective in CO_2 elimination when applied in late expiration. Our results suggest that TGE can be added to reduce TGI-induced EELV increase while retaining the distal and proximal CO_2 elimination effects of antegrade TGI. However, we note that this study was not conducted under conditions of disordered physiology, which is an important qualification of the results and implications. Further investigation of phasic TGI and TGE in disease analogs and with other ventilation patterns is clearly needed before TGI/TGE can be recommended for the clinical setting.

REFERENCES

1. Ravenscraft S, Burke WC, Nahum A, Adams AB, Nakos G, Marcy TW, Marini JJ. Tracheal gas insufflation augments CO_2 clearance during mechanical ventilation. Am Rev Respir Dis 1993;148(2):345–351.
2. Burke WC, Nahum A, Ravenscraft SA, Nakos G, Adams AB, Marcy TW, Marini JJ. Modes of tracheal gas insufflation: comparison of continuous and phase-specific gas injection in normal dogs. Am Rev Respir Dis 1993;148(3):562–568.
3. Slutsky AS, Menon AS. Catheter position and blood gases during constant flow ventilation. J Appl Physiol 1987;62(2):513–519.
4. Slutsky AS, Watson J, Leith D, Brown R. Tracheal insufflation of O_2 (TRIO) at low flow rates sustains life for several hours. Anesthesiology 1985;63(3):278–286.
5. Stresemann E, Votteri BA, Sattler FP. Washout of anatomic dead space for alveolar hypoventilation. Respiration 1969;26(6):425–434.
6. Stresemann E. Washout of anatomical dead space: design of method and experimental study using external dead space. Respiration 1968;25(4):281–291.
7. Ravenscraft SA, Shapiro RS, Nahum A, Burke WC, Adams AB, Nakos G, Marini JJ. Tracheal gas insufflation: catheter effectiveness determined by expiratory flush volume. Am J Respir Crit Care Med 1996;153(6 Pt 1):1817–1824.
8. Nakos G, Zakinthinos S, Kotanidou A, Tsagaris H, Roussos C. Tracheal gas insufflation reduces the tidal volume while P_{ACO_2} is maintained constant. Intensive Care Med 1994;20(6):407–413.
9. Kuo PH, Wu HD, Yu CJ, Yang SC, Lai YL, Yang PC. Efficacy of tracheal gas insufflation in acute respiratory distress syndrome with permissive hypercapnia. Am J Respir Crit Care Med 1996;154(3 Pt 1):612–616.
10. Hoyt JD, Marini JJ, Nahum A. Effect of tracheal gas insufflation on demand valve triggering and total work during continuous positive airway pressure ventilation. Chest 1996;110(3):775–783.
11. Nahum A, Burke WC, Ravenscraft SA, Marcy TW, Adams AB, Crooke PS, Marini JJ. Lung mechanics and gas exchange during pressure-control ventilation in dogs. Augmentation of CO_2 elimina-

tion by an intratracheal catheter. Am Rev Respir Dis 1992;146(4): 965–973.

12. Nahum A, Ravenscraft SA, Nakos G, Burke WC, Adams AB, Marcy TW, Marini JJ. Tracheal gas insufflation during pressure-control ventilation: effect of catheter position, diameter, and flow rate. Am Rev Respir Dis 1992;146(6):1411–1418.
13. Maloney IV, Elam JO, Handford SW, Balla GA, Army US, Eastwood DW, Brown ES, Ten Pas RH. Importance of negative pressure phase in mechanical respirators. JAMA 1953;152(3):212–216.
14. Beaver RA. Pressure respirators: the application of a negative phase. Lancet 1955;Sept 17:588–589.
15. Motley HL, Cournand A, Werko L, Dresdale DT, Himmelstein A, Richards DW. Intermittent positive pressure breathing; a means of administrating artificial respiration in man. JAMA 1948;137(4):370–382.
16. DuPuis YG. Ventilators: theory and clinical application. St Louis: CV Mosby; 1986:180–181.
17. Takahashi T, Bugedo G, Adams AB, Marini JJ. A comparison of augmented CO_2 elimination from a lung model by tracheal gas insufflation (TGI), negative expiratory airway pressure (NEAP) and TGI-NEAP combined (abstract). Am J Respir Crit Care Med 1997; 155(4):A528.
18. Takahashi T, Bugedo G, Adams AB, Marini JJ. The effect of tracheal gas insufflation (TGI), negative expiratory airway pressure (NEAP) and TGI-NEAP combined on autoPEEP in a lung model (abstract). Am J Respir Crit Care Med 1997;155(4):A529.
19. Belghith M, Fierobe L, Brunet F, Monchi M, Mira JP. Is tracheal gas insufflation an alternative to extrapulmonary gas exchangers in severe ARDS? Chest 1995;107(5):1416–1419.
20. Imanaka H, Kacmarek RM, Ritz R, Hess D. Tracheal gas insufflation—pressure control versus volume control ventilation: a lung model study. Am J Respir Crit Care Med 1996;153(3):1019–1024.
21. Nahum A, Ravenscraft SA, Nakos G, Adams AB, Burke WC, Marini JJ. Effect of catheter flow direction on CO_2 removal during tracheal gas insufflation in dogs. J Appl Physiol 1993;75(3):1238–1246.
22. Nahum A, Ravenscraft SA, Adams AB, Marini JJ. Distal effects of tracheal gas insufflation: changes with catheter position and oleic acid lung injury. J Appl Physiol 1996;81(3):1121–1127.
23. Kolobow T, Powers T, Mandava S, Aprigliano M, Kawaguchi A, Tsuno K, Mueller E. Intratracheal pulmonary ventilation (ITPV): control of positive end-expiratory pressure at the level of carina through the use of a novel ITPV catheter design. Anesth Analg 1994;78(3):455–461.
24. Imanaka H, Kirmse M, Mang H, Hess D, Kacmarek RM. Expiratory phase tracheal gas insufflation and pressure control in sheep with permissive hypercapnia. Am J Respir Crit Care Med 1999;159(1): 49–54.
25. Adams AB. Tracheal gas insufflation (TGI). Respir Care 1996;41(4): 285–291; discussion: 292–293.
26. Ravenscraft SA. Tracheal gas insufflation: adjunct to conventional mechanical ventilation. Respir Care 1996;41(2):105–111.
27. Bergofsky EH, Hurewitz AN. Airway insufflation: physiologic effects on acute and chronic gas exchange in humans. Am Rev Respir Dis 1989;140(4):885–890.
28. Hurewitz AN, Bergofsky EH, Vomero E. Airway insufflation: increasing flow rates progressively reduce dead space in respiratory failure. Am Rev Respir Dis 1991;144(6):1229–1233.
29. Couser JI Jr, Make BJ. Transtracheal oxygen decreases inspired minute ventilation. Am Rev Respir Dis 1989;139(3):627–631.
30. Benditt JO, Pollock M, Roa J, Celli B. Transtracheal delivery of gas decreases the oxygen cost of breathing. Am Rev Respir Dis 1993; 147(5):1207–1210.
31. Danan C, Dassieu G, Janaud JC, Brochard L. Efficacy of dead-space washout in mechanically ventilated premature newborns. Am J Respir Crit Care Med 1996;153(5):1571–1576.
32. Dassieu G, Brochard L, Agudze E, Patkai J, Janaud JC, Danan C. Continuous tracheal gas insufflation enables a volume reduction strategy in hyaline membrane disease: technical aspects and clinical results. Intensive Care Med 1998;24(10):1076–1082.

A Bench Study Comparison of Demand Oxygen Delivery Systems and Continuous Flow Oxygen

Peter L Bliss BME, Robert W McCoy RRT BSM, and Alexander B Adams RRT MPH

BACKGROUND: Currently available methods for conserving oxygen include transtracheal catheters, reservoir cannulae, and demand oxygen delivery systems (DODS). DODS do not deliver oxygen during exhalation, and most models do not deliver oxygen during portions of the inspiratory cycle. To determine selected performance differences between DODS models and continuous flow oxygen (CFO), we conducted a bench model evaluation of 7 DODS models and CFO. METHODS: A bench model was constructed to simulate a nose, airway, and alveolar chamber. A linked ventilator drove the alveolar chamber to generate 3 respiratory patterns, at frequencies of 15, 20, and 26 per minute. DODS and CFO were tested at settings of 1, 2, 4, and 6 L/min. The fraction of inspired oxygen (F_{IO_2}) in the alveolar chamber was measured for each condition. Oxygen use efficiency (OUE) was calculated for each device by determining oxygen in the alveolar chamber/oxygen used by the device. RESULTS: The DODS models differed with regard to flow delivery profile. Supplemental oxygen (F_{IO_2} increase over 21%) for CFO declined markedly (43%) with increasing frequency, and was better maintained for the DODS models (average of 10% decrease). DODS that deliver an early inspiratory pulse tended to maintain F_{IO_2} with increases in frequency and to be more efficient with oxygen use. OUE was 21% for CFO, and ranged from 42% to 100% for DODS models. CONCLUSIONS: DODS models were not equivalent to CFO or to each other in F_{IO_2} or OUE. DODS conserve oxygen and may maintain F_{IO_2} better than CFO when respiratory frequency increases. DODS models differ and may require setting adjustments to achieve equivalent F_{IO_2} to CFO or to other models. To optimally customize oxygen therapy to the patient, the DODS model or CFO should be set to meet adequate saturation goals (ie, > 90%) under conditions of usual use, including rest and exercise. [Respir Care 1999;44(8):925–931] *Key words: demand oxygen delivery systems, continuous flow oxygen, oxygen use efficiency.*

Introduction

Although most oxygen therapy is administered as continuous flow oxygen (CFO), 3 methods of oxygen conservation have come into use. Transtracheal catheters deliver oxygen directly into the trachea, requiring approximately half the oxygen of a nasal cannula system to provide equivalent oxygen saturation.[1] Reservoir cannulae have a reservoir near the nose that fills with oxygen during exhalation,[1,2] and the reserved oxygen is inhaled at the onset of the next inhalation. A reduced flow setting thus conserves a significant amount of oxygen. Demand oxygen delivery systems (DODS) interrupt the flow of oxygen during exhalation, when the oxygen flow is otherwise mostly wasted. DODS either pulse oxygen early in inhalation or provide oxygen flow throughout inhalation. DODS use standard or modified nasal cannulae, and are set with "CFO equivalent" settings. The use-time of portable devices can then be extended, and/or smaller, lighter devices can be used. Transtracheal catheters and reservoir cannulae have found relatively low acceptance compared to DODS. First available in the 1980s, there were 18,000 DODS in use in 1994,[2] and approximately 80,000 units (industry estimate) were put into service in the United States in 1998.

The various DODS models interrupt exhalation-phase oxygen flow by different methods, thus producing different flow profiles. DODS flow is not the same as CFO, so controversy exists as to the "equivalence" of DODS and

---

Peter L Bliss BME, Robert W McCoy RRT BSM, and Alexander B Adams RRT MPH are affiliated with Regions Hospital, Pulmonary Research, 640 Jackson Street, St Paul, Minnesota.

Mr Bliss presented a version of this paper at the American Association for Respiratory Care Open Forum during the 44th International Respiratory Congress, November 7–10, 1998, in Atlanta, Georgia.

Correspondence: Peter L Bliss BME, 3953 W 143rd St, Savage MN 55378. E-mail: pbliss@inspiredrc.com.

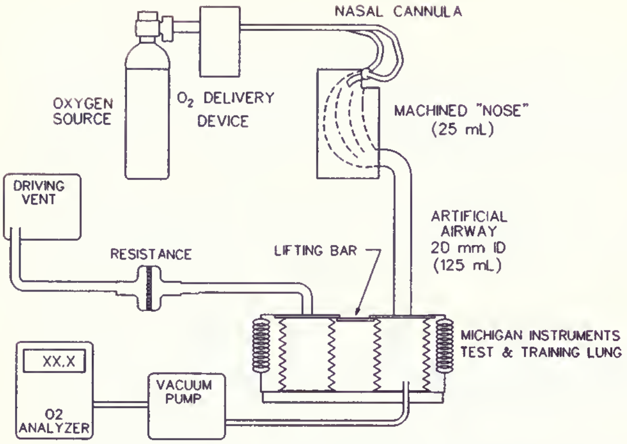


Fig. 1. Schematic of the demand oxygen delivery system/continuous flow oxygen (DODS/CFO) evaluation system. ID = internal diameter.

CFO settings. Each DODS manufacturer uses algorithms based on certain assumed breathing patterns to create CFO equivalent settings for their model. While DODS may function similarly to CFO under the assumed conditions, they may deviate from equivalence with other breathing patterns. Thus, clinical studies have shown DODS to produce similar results to CFO,[1,3–6] but there are studies that report differences.[7–10]

Several difficulties are apparent in comparing DODS to CFO. The variability and lability in patients' breathing patterns make it difficult to control for and compare possible differences in oxygenation or exercise tolerance between DODS and CFO. Also, arterial oxygen saturation, which is often the outcome variable in comparison studies, shows small changes for large changes or differences in F_{IO_2} or arterial oxygen tension (P_{aO_2}). In studies comparing DODS with CFO, the initial CFO setting might be titrated with little or no effort to optimize it. It is possible that in studies in which oxygen savings over CFO were reported, a small reduction in the CFO prescription may have also realized oxygen savings without adverse effect on arterial oxygen saturation. Similarly, in studies that found DODS to be inferior to CFO at exercise, settings equivalent to CFO were used.[7–10] It is possible that simply increasing the DODS setting would have created equal or better results while still saving oxygen.

Given the delivery differences between DODS and CFO, "equivalent" settings present difficulties in clinical comparisons. We devised a test lung model constructed to evaluate differences between DODS and CFO during three simulated breathing patterns. Differences found in a controlled setting, without anatomic, physiologic or other clinical variables, might help to explain clinical nonequivalence and might enable more knowledgeable setting of DODS to meet oxygenation goals and realize oxygen savings.

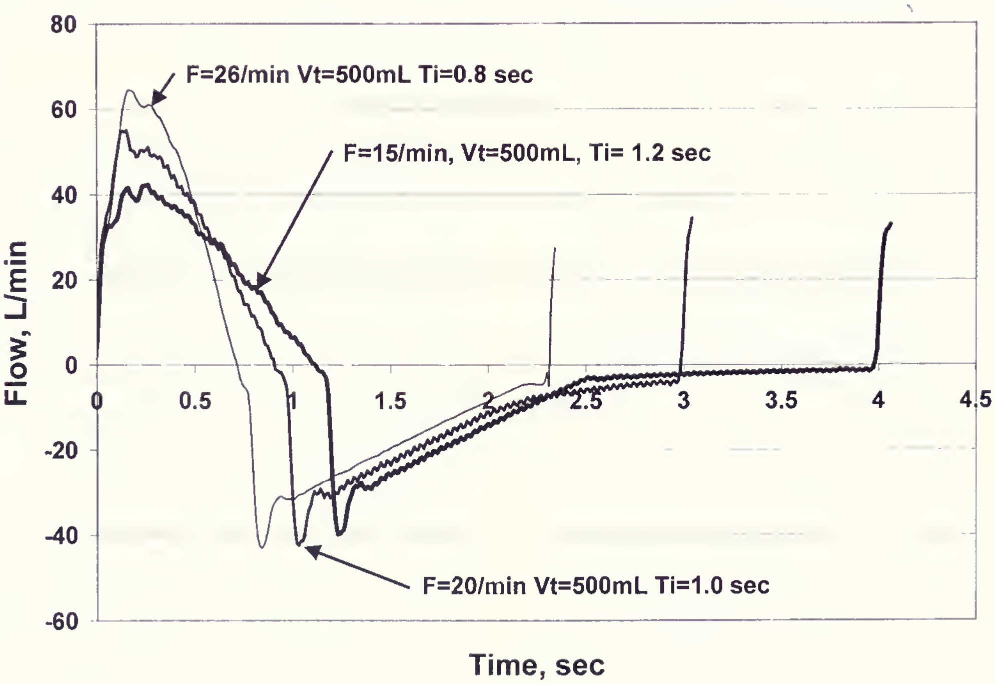


Fig. 2. Flow waveforms for low (f = 15/min), medium (f = 20/min), and high (f = 26/min) respiratory frequency breathing patterns. Vt = tidal volume. Ti = inspiratory time.

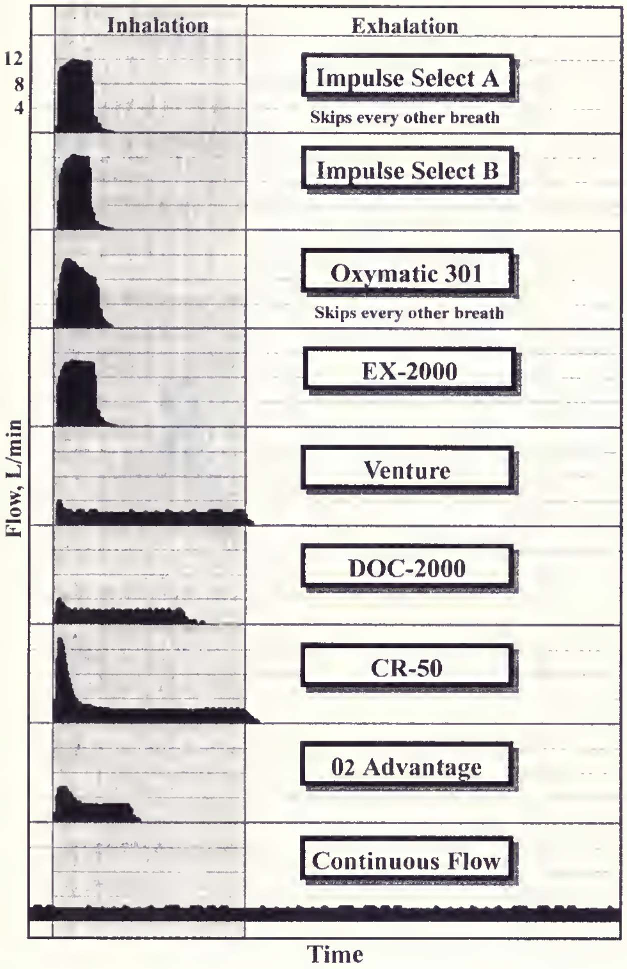


Fig. 3. Oxygen delivery flow profile for each demand oxygen delivery system (DODS) model, measured at a setting of 2, respiratory rate = 20/min, and inspiratory-expiratory ratio (I-E) = 1:2. The Impulse Select model DODS has 2 modes (A and B).

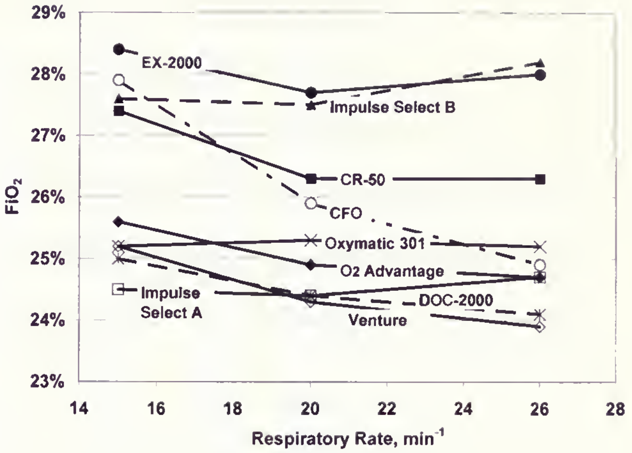


Fig. 4. Fraction of inspired oxygen (F_{IO_2}) measured for each demand oxygen delivery system (DODS) model and continuous flow oxygen (CFO) during the 3 breathing patterns shown in Fig. 2, as driven by the linked ventilator. Continuous flow oxygen (CFO) was set at 2 L/min, and the DODS models were set at 2.

Equipment and Methods

Seven currently available DODS models (described in Appendix 1), and CFO, were evaluated using a mechanical lung model. The model was constructed to simulate a nose, conducting airways, and an alveolar chamber (Fig. 1). The conducting airways and nose represented a dead space volume of 150 mL. The apparatus was connected to a "respirating" limb of a test lung (Michigan Instruments, Grand Rapids, Michigan), driven by a linked ventilator (LP-6, Aequitron Medical, Plymouth, Minnesota). Three respiratory patterns were generated by the ventilator, representing low (f = 15/min), medium (f = 20/min), and high (f = 26/min) respiratory rate conditions, with tidal volume (V_T) = 500 mL, and inspiratory-expiratory ratio = 1:2 (Fig. 2). Prior to the F_{IO_2} testing, the oxygen flow profile output from the DODS models were measured by an electronic flow meter (model 4040, TSI Inc, St Paul, Minnesota). Oxygen used was also measured by integrating the flow, using the TSI flow meter. F_{IO_2} in the alveolar chamber was measured by an oxygen analyzer (model OM-25A, Ceramatec Inc, Salt Lake City, Utah) for each breathing pattern, at 1, 2, 4, and 6 L/min settings for each DODS and for CFO.

Since oxygen use and F_{IO_2} vary between devices, an oxygen use efficiency (OUE) calculation was developed to evaluate the ability of each DODS model to both deliver and conserve oxygen. The OUE considers the amount of supplemental oxygen in the alveolar chamber (as measured via F_{IO_2}) relative to the amount of oxygen used from the source.

$$\text{OUE} = \frac{\text{supplemental oxygen in test lung}}{\text{oxygen used from source}}$$

$$\text{OUE} = \frac{(F_{IO_2} - 0.21) \times (V_T - V_D) \times 1/0.79}{V_{O_2}}$$

Where V_T = tidal volume (mL), V_D = dead space volume (mL), V_{O_2} = oxygen used from source per breath (mL), and F_{IO_2} = oxygen concentration measured in test lung.

Appendix 2 shows the derivation of the OUE calculation. The overall OUE was calculated by averaging the values for each of the 3 breathing patterns and the settings of 1, 2, and 4 L/min (setting 6 was not included because not all models have setting 6).

Results

The oxygen delivery flow profiles are shown in Figure 3. There were clear differences in flow profiles between

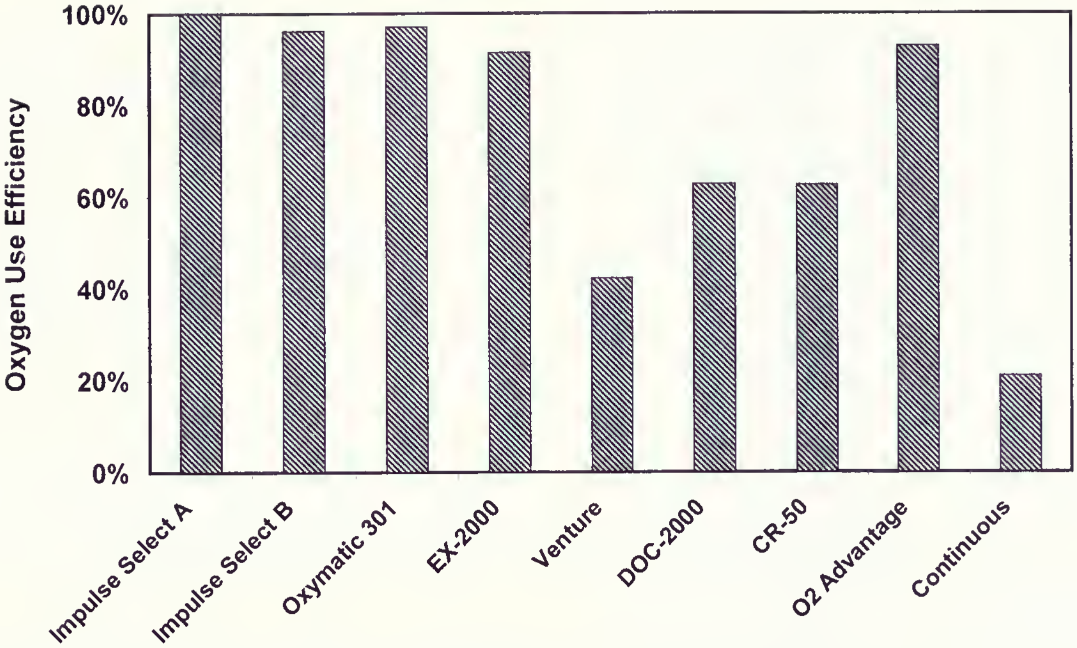


Fig. 5. Calculated oxygen use efficiency for 7 demand oxygen delivery system models and continuous flow oxygen.

DODS models; therefore, the models were identified as three types: pulse, demand and hybrid. The Impulse Select, Oxymatic 301, and EX-2000 models deliver an early inspiratory *pulse* of oxygen at a flow rate higher than the "equivalent" rate (setting in L/min). The Venture and DOC-2000 *demand* models deliver flow at the "equivalent" rate during most of the inhalation phase, as if in response to inspiratory demand. The CR-50 and O2 Advantage models are *hybrid* models with an early inspiratory pulse and a subsequent "equivalent" flow period.

Figure 4 shows the F_{IO_2} measurements for the 2 L/min setting. At settings of 1, 4, and 6 L/min, the devices displayed similar F_{IO_2} response to changing breathing patterns. There were differences between CFO and the DODS models in absolute values of F_{IO_2} and in patterns of response to increasing respiratory frequency. The F_{IO_2} values for CFO declined markedly with increasing frequency, while F_{IO_2} for the DODS models was maintained or declined slightly. Supplemental oxygen (F_{IO_2} increase over 21%) decreased 43% from low to high respiratory frequency for CFO. Pulse DODS models increased an average of 2%, demand models decreased 27%, and hybrid models decreased 18%.

Figure 5 shows the calculated OUE values. All of the DODS models tested provided more efficient oxygen delivery than CFO. The DODS displayed a wide range of efficiency values, and the pulse DODS models tended to achieve higher efficiency values than the demand or hybrid models.

Discussion

In this controlled-setting comparison of DODS models and CFO we found differences in absolute values of F_{IO_2} and patterns of response to increasing respiratory rate. Two previous studies comparing DODS models have found performance differences between models.[8,10] We now report differences between models currently available that associate their flow delivery profiles with oxygen delivery and oxygen conservation. This can be explained by 3 factors: pooling, dilution, and timing of oxygen delivery.

Pooling

The higher F_{IO_2} delivered by CFO at the lower respiratory frequency can be explained by an anatomic reservoir, allowing pooling of oxygen from the end of the last exhalation. This pooling would occur if a potential chamber (nose) exists and end-expiratory flow from the patient has ended or is negligible. End-expiratory flow would flush the reservoir. In our model, as respiratory frequency increased, this effect diminished, since expiratory flow extends toward end-exhalation as expiratory time shortens. Demand type DODS delivered lower F_{IO_2} than CFO at low respiratory frequency, and nearly the same at high frequency. The pooling effect would explain this.

Dilution

With increasing respiratory frequency, the increase in minute ventilation dilutes the oxygen dose from CFO. The pulse DODS models deliver the same volume of oxygen per breath regardless of frequency. This could explain the flatter F_{IO_2} response to increasing respiratory frequency with the pulse DODS models. Shortened inspiratory time might reduce dosing with the demand and hybrid models. The response of F_{IO_2} to increasing frequency is not as well maintained with the demand and hybrid models as with the pulse models.

Timing of Oxygen Delivery

The observed efficiency was also partly determined by the timing of the oxygen delivery. Assuming bulk gas flow, the final 30% of an inspiratory volume would occupy dead space (150 mL dead space, 500 mL V_T). If oxygen is delivered early in the inspiration, it is more likely to reach the alveoli, which would explain the higher efficiency of the early delivery pulse type DODS. Late inspiratory oxygen may just occupy proximal, dead space regions, so a demand type DODS that delivers oxygen during the later portion of inhalation would be less efficient than a pulse type, which delivers only during the early portion of inhalation. There are 2 hybrid devices: one that delivers throughout inhalation is less efficient than one that shortens delivery to a portion of inhalation. The differences in efficiency values are striking. The values imply that approximately 20% of the CFO reaches the alveolar chamber, while with some of the DODS models, almost 100% of the oxygen reaches the alveolar chamber.

There are several limitations of this bench study that suggest caution in extrapolating the findings to the clinical setting. First, this study does not account for the wide variability in breathing patterns. Only 3 breathing patterns were tested at 4 flow settings—hardly the full range of clinical use. The breathing patterns tested are not necessarily representative of other conditions such as sleep, vigorous exercise, or restrictive lung disease. For example, triggering-sensitivity of DODS may be an important issue when breathing is shallow. Device triggering sensitivity was not quantified in this study. Second, our lung model represents only some mechanical aspects of ventilation. Flow through a machined nose and smooth bore tubing may not produce the gas mixing characteristics of the human airway. Furthermore, the model cannot represent the distribution of oxygen throughout the lungs or its adsorption by the blood. Ventilation/perfusion relationships and gas distribution differ between patients and bear on oxygen delivery. Finally, since no oxygen was consumed in our setup, the actual F_{IO_2} measurements are artificially high and cannot be used for clinical comparison. Nevertheless, the model highlights differences between DODS devices and CFO. It is clear that further investigation of DODS is necessary, particularly in the clinical setting.

Conclusions

As use of DODS spreads, a thorough understanding of their operation and appropriate application becomes increasingly important. In this bench study comparison of DODS models and CFO, we found differences that may be relevant to clinical practice. DODS models were not equivalent to CFO or to each other in F_{IO_2} delivery or OUE.

All 7 DODS models conserved oxygen (compared to CFO) and maintained a more constant F_{IO_2} with increasing respiratory frequency than did CFO. The pulse DODS models tended to conserve more oxygen and maintain a more constant F_{IO_2} than the demand and hybrid DODS models.

Further testing of DODS should include titration of settings under a range of conditions to attain adequate saturation while evaluating oxygen savings. To customize oxygen therapy to the patient, the DODS model or CFO should be set to meet adequate saturation goals (ie, > 90%) under conditions of usual use, including rest and exercise.

REFERENCES

1. Yaeger ES, Goodman S, Hoddes E, Christopher KL. Oxygen therapy using pulse and continuous flow with a transtracheal catheter and nasal cannula. Chest 1994;106(3):854–860.
2. Barker AF, Burgher LW, Plummer AL. Oxygen conserving methods for adults (review). Chest 1994;105(1):248–252.
3. Yuan LC, Jun Z, Min LP. Clinical evaluation of pulse-dose and continuous-flow oxygen delivery. Respir Care 1995;40(8):811–814.
4. Tiep BL, Carter R, Nicotra B, Berry J, Phillips RE, Otsap B. Demand oxygen delivery during exercise. Chest 1987;91(1):15–20.
5. Carter R, Tashkin D, Djahed B, Hathaway E, Nicotra MB, Tiep BL. Demand oxygen delivery for patients with restrictive lung disease. Chest 1989:96(6):1307–1311.
6. Garrod R, Bestall JC, Paul E, Wedzicha JA. Evaluation of pulsed dose oxygen delivery during exercise in patients with severe chronic obstructive pulmonary disease. Thorax 1999;54(3):242–244.
7. Hagarty EM, Skorodin MS, Langbein WE, Hultman CI, Jessen JA, Maki KC. Comparison of three oxygen delivery systems during exercise in hypoxemic patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1997;155(3):893–898.
8. Braun SR, Spratt G, Scott GC, Ellersieck M. Comparison of six oxygen delivery systems for COPD patients at rest and during exercise. Chest 1992;102(3):694–698.
9. Roberts CM, Bell J, Wedzicha JA. Comparison of the efficacy of a demand oxygen delivery system with continuous low flow oxygen in subjects with stable COPD and severe oxygen desaturation on walking. Thorax 1996;51(8):831–834.
10. Dawson AD, Elias DJ, Keesling L, Averell P. Comparison of oxygen conserving devices during exercise in hypoxemic patients with interstitial lung disease (abstract). Am J Respir Crit Care Med 1998; 157(3):A95.

Appendix I

Devices Tested

Unless otherwise noted, all devices use a standard nasal cannula and operate on alkaline batteries.

Impulse Select. Airsep Corporation (www.airsep.com), Buffalo, New York. Airsep manufactures 2 demand oxygen delivery systems: the *Impulse* and the *Impulse Select.* We tested the *Impulse Select.* In operation mode A, the device operates similar to the CHAD *Oxymatic 301* (see below), delivering a 35 mL pulse at every breath when set at 4, skipping breaths at lower settings. At settings 5 and 6, the pulse is larger. In operation mode B, the device operates similar to the DeVilbiss *EX2000*, delivering fixed flow rate (12 L/min) pulses of longer duration with increasing setting. Includes built-in high pressure regulator for use with cylinder. The *Impulse* model operates similar to the CHAD *Oxymatic 301.*

Oxymatic 301. CHAD Therapeutics Inc (www.chadtherapeutics.com), Chatsworth, California. Delivers a fixed volume pulse (approximately 38 mL) of oxygen at the onset of inhalation. At setting 4, delivers every breath. At settings 3, 2, and 1, delivers on 3 of 4, every other, and every fourth breath, respectively. Uses any 20 psi oxygen source with a 4 L/min flow control.

EX-2000. DeVilbiss, a division of Sunrise Medical Inc (www.sunrisemedical.com), Somerset, Pennsylvania. Integrated high pressure regulator and conserving device that fits around the post valve of a high pressure cylinder. Delivers a pulse at 10 L/min at the onset of each inhalation. On time is varied from 0.050 to 0.6 seconds, depending on the setting. DeVilbiss also manufactures a liquid oxygen portable system with a similar demand oxygen delivery system built in.

Venture. Invacare Corporation (www.invacare.com), Elyria, Ohio. Connects to a standard fixed orifice regulator or liquid system. Delivers externally set "equivalent" flow rate for 1.0 seconds beginning at the onset of each inhalation. Rechargeable internal battery or AC adapter.

CR-50. Nellcor Puritan Bennett Corporation, a division of Mallinckrodt Inc (www.nellcorpb.com), St Charles, Missouri. Integrated high pressure regulator, flow control and demand valve that mounts directly to a high pressure cylinder. Delivers a 12 mL bolus at the onset of each inhalation, then continues to deliver at the set flow rate (1–6 L/min) until the end of inhalation. Uses dual lumen cannula. Settings of 0.25, 0.50, and 0.75 L/min are automatically delivered in a continuous flow oxygen mode. Pneumatic (no battery). Nellcor Puritan Bennett also manufactures a liquid oxygen portable system with a similar demand oxygen delivery system built in.

DOC-2000. Transtracheal Systems (www.transtracheal.com), Englewood, Colorado. Connects to a standard fixed orifice regulator or liquid system. Delivers externally set "equivalent" flow rate for 0.7 seconds beginning at the onset of each inhalation. Similar in function to the Invacare *Venture. DOC-2000* is the only device that delivers continuous flow oxygen in the event of power failure, battery depletion, or apnea. Rechargeable, replaceable external battery or AC adapter.

O2 Advantage. Western Medica (www.westernmedica.com), Westlake, Ohio. Connects to a standard fixed orifice regulator or liquid system. Delivers a small bolus at the onset of inhalation and continues to flow at the externally set "equivalent" flow for a set portion of inhalation. Device can be set with internal switches to deliver for 33, 40, 50, or 70% of measured inspiratory time. Uses dual lumen cannula. Factory setting of 40% used in this study.

Appendix 2

Oxygen Use Efficiency Calculation

V_T	Tidal Volume (in mL)
V_D	Dead Space Volume (in mL)
V_{O_2}	Volume of Oxygen Used from Source (mL per Breath)
V_{O_2T}	Volume (in mL) of Oxygen Contained in Tidal Volume
V_{O_2L}	Volume of Oxygen Entering Alveolar Chamber (mL per Breath)
V_{AIR}	Volume of Air Entering Alveolar Chamber (mL per Breath)
F_{IO_2}	Fraction of Inspired Oxygen Measured

Define:

$$\text{Oxygen Use Efficiency} = V_{O_2L}/V_{O_2} \qquad (1)$$

Mass balance assumptions:

$$V_T = V_{AIR} + V_{O_2L} + V_D \qquad (2)$$

$$V_{O_2T} = V_T \times F_{IO_2} = 0.21 \times V_{AIR} + V_D \times F_{IO_2} + V_{O_2L} \qquad (3)$$

Solve equation (3) for V_{O_2L}:

$$V_{O_2L} = V_T \times F_{IO_2} - 0.21 \times V_{AIR} - V_D \times F_{IO_2} \qquad (4)$$

Solve (2) for V_{AIR}. Substitute into equation (4) and simplify:

$$V_{O_2L} = V_T \times F_{IO_2} - 0.21 \times (V_T - V_D - V_{O_2L}) - V_D \times F_{IO_2}$$

$$V_{O_2L} - 0.21 \times V_{O_2L} = (V_T - V_D) \times F_{IO_2} - 0.21 \times (V_T - V_D)$$

$$V_{O_2L} = (V_T - V_D) \times (F_{IO_2} - 0.21) \times {}^{1}/_{0.79} \qquad (5)$$

Substitute equation (5) into equation (1):

$$\text{Oxygen Use Efficiency} = \frac{(V_T - V_D) \times (F_{IO_2} - 0.21) \times {}^{1}/_{0.79}}{V_{O_2}}$$

Clinical Validation of the Functional Performance Inventory in Patients with Chronic Obstructive Pulmonary Disease

Nancy Kline Leidy PhD and Ann R Knebel DNSc RN

OBJECTIVE: Examine the clinical validity of the Functional Performance Inventory (FPI), a new functional outcome indicator for patients with chronic obstructive pulmonary disease (COPD). METHODS: Subjects with COPD were recruited from an asthma and allergy clinic where they completed the FPI, Functional Status Questionnaire (FSQ), Duke Activity Status Index (DASI), pulmonary function tests (PFT) and 12-minute walk distance (12-MWD) test. Subjects wore a wrist actigraph to monitor physical movement over a 3-day mid-week period while they recorded their physical activities, activity difficulty, and symptoms (dyspnea, fatigue) using the NIH Activity Record (ACTRE). PATIENTS: Twenty-two patients (13 men, 9 women) participated in the study. Mean (± SD) age of the sample was 64 ± 10 years; forced expiratory volume in the first second (FEV_1) = 1.0 ± 0.46 L; percent of predicted FEV_1 = 37.30% ± 3.99%; and 12-MWD = 1683 ± 872 ft. RESULTS: FPI total score correlated significantly ($p < 0.05$) with percent of predicted FEV_1 ($r = 0.55$), 12-MWD ($r = 0.52$), diary data for dyspnea ($r = -0.59$), fatigue ($r = -0.62$) and difficulty with activity ($r = -0.71$), and FSQ basic ($r = 0.61$) and intermediate ($r = 0.70$) activities of daily living. Correlations with the DASI ($r = 0.43$) and actigraph ($r = 0.26$) were moderate, but not statistically significant. The household maintenance subscale correlated ($p < 0.05$) with all of the validation measures ($r = 0.52$ to 0.81), including the activity monitor ($r = 0.52$). CONCLUSIONS: The results suggest the FPI is a valid instrument for evaluating subjective performance in COPD patients. [Respir Care 1999;44(8):932–939] *Key words: obstructive lung disease, functional performance inventory, functional status, activities of daily living.*

Functional status is an important indicator of the impact of disease on daily life and can serve as a primary outcome in evaluating the effectiveness of treatment. A comprehensive evaluation of functional status includes assessment of both the patient's functional capacity (ie, his or her *potential* to participate in daily activity based on their physical or psychological health) and his or her functional performance (ie, the degree of difficulty with which activities are *actually performed* on a day-to-day basis).[1,2] Clinical observation and empirical evidence suggest that chronic obstructive pulmonary disease (COPD) constrains daily activities, but the absence of a comprehensive, condition-targeted, patient-based assessment tool has limited our ability to profile these performance difficulties, target interventions toward specific domains of activity, and evaluate treatment in terms of performance outcomes.

Attempts have been made to use the generic Sickness Impact Profile (SIP) to evaluate functional performance in COPD patients, but the SIP confounds physical and psychological symptoms with activity, making it impossible to isolate effects. The SIP has also been insensitive to change in the COPD population.[3–6] Instruments such as the Functional Status Questionnaire (FSQ), Duke Activity Status Index (DASI), Index of Activities of Daily Living, Barthel Index, and the PULSES (physical, upper extremities, lower extremities, sensory, excretory, social support [physical profile]) focus heavily on activities of daily living and are limited in (or exclude altogether) the performance of recreational or social activities.[7–11] The Karnofsky Performance Status Scale must be administered by a

---

Nancy Kline Leidy PhD is affiliated with the Center for Health Outcomes Research, MEDTAP International, and the Division of Intramural Research, National Institutes of Health, Bethesda, Maryland. Ann R Knebel DNSc RN is affiliated with the Clinical Center Nursing Department of the National Institutes of Health, Bethesda, Maryland.

This study was funded by the Division of Intramural Research, National Institute of Nursing Research, National Institutes of Health, Protocol Z01 NR00008–01 HRHI, and conducted at the Johns Hopkins Asthma and Allergy Center, Baltimore, Maryland.

Correspondence: Nancy Kline Leidy, Center for Health Outcomes Research, MEDTAP International Inc, 7101 Wisconsin Avenue, Suite 600, Bethesda MD 20814. Email: Leidy@MEDTAP.com.

professional, which increases costs, complicates designs, and makes large surveys or outcome studies virtually impossible. In addition, the Karnofsky Performance Status Scale was intended for use with cancer patients, which raises questions about its utility and efficacy across patient populations.[12]

Two measures of functional status specific to the COPD population have been developed to date: the Pulmonary Functional Status and Dyspnea Questionnaire (PFSDQ)[13,14] and the Pulmonary Functional Status Scale (PFSS).[15] The development and evaluation of these instruments are well documented, and both have shown evidence of reliability and validity as functional status measures. However, both have limitations in light of the multidimensional nature of functional status and the need to isolate and operationalize functional performance across multiple domains.[1,2] The intent of the 164-item PFSDQ and the 40-item PFSDQ-modified, for example, is to measure both dyspnea intensity and change in the patient's ability to perform daily activities after the onset of disease, rather than to profile current level of performance. The 35-item PFSS describes patient functional status along 3 dimensions: daily activities/social functioning, psychological functioning, and sexual functioning, with specific domains of activity subsumed within these dimensions. Activities related to body care, household maintenance, and recreation, for example, are included in the daily activities/social functioning score. This limits the user's ability to identify specific domains of activity that are problematic or that are responding to treatment.

The Functional Performance Inventory (FPI) was designed to evaluate the COPD patient's subjective perception of functional performance across 6 domains of activity: body care, household maintenance, physical exercise, recreation, spiritual activities, and social activities. Items and structure of the instrument were based on an analytical framework, in-depth review of the literature, qualitative interviews with COPD patients, and input from respiratory clinicians.[1,2,16,17] Internal consistency reliability, reproducibility and construct, concurrent and discriminant validity of the instrument were examined in a cross-sectional survey of 154 patients with COPD and a sub-sample of 40 relatives.[16]

The present study advances previous research on the validity of the FPI by examining the clinical validity of the instrument, detailing its performance in a small sample of COPD patients. Specifically, this study explores the degree to which the FPI relates to (1) disease severity (ie, elements of capacity that constrain performance: forced expiratory volume in 1 second as a percentage of predicted value [FEV_1 percent of predicted], and 12-minute walk distance [12-MWD]); (2) patient self-report of activity-related dyspnea, fatigue and difficulty, gathered through a daily diary, the National Institutes of Health (NIH) Activity Record (ACTRE);[18,19] (3) activity levels recorded with an electronic motion detector, the Dual-Mode Mini-Motionlogger actigraph (Ambulatory Monitoring Inc, Ardsley, New York), and (4) two alternative subjective measures of activity that were consistent with the underlying framework[1,2] but less comprehensive than the FPI—the Duke Activity Status Index (DASI)[8] and the FSQ Activities of Daily Living (ADL) and Intermediate Activities of Daily Living (IADL) scales.[9]

Methods

Subjects

Twenty-three subjects, recruited through the Johns Hopkins Asthma and Allergy Center, participated in the study. Criteria for eligibility were medical diagnosis of COPD, chronic bronchitis, or emphysema; an FEV_1 percent of predicted $\leq 70\%$; freedom from other conditions that adversely affect performance (eg, obesity, use of a cane or walker, severe arthritis); the ability and willingness to come to the clinic for pulmonary function tests (PFTs) and 12-MWD testing; and ability to wear and interest in wearing an actigraph and recording activities in a diary for 3 days.

Measures

The Functional Performance Inventory (FPI) asks subjects to respond to each of 65 activities, identifying those they choose to perform and rating the ease/difficulty with which they are performed. The response scale ranges from 0 to 4, with an option to indicate that the activity is not performed for reasons other than health. Items include activities COPD patients often find difficult, such as washing hair, caring for feet, vacuuming, changing sheets, mowing the lawn, getting in and out of a car, short walks around the neighborhood or mall, talking on the telephone, and overnight travel. Six subscales comprise the FPI: body care (9 items), household maintenance (21 items), physical exercise (7 items), recreation (11 items), spiritual activities (5 items), and social activities (12 items). Higher scores on the FPI indicate higher levels of performance. The internal consistency reliability, α coefficient, for the FPI total score in this sample was 0.98. The α coefficients for the 6 subscales were as follows: body care, 0.87; household maintenance, 0.90; physical exercise, 0.78; recreation, 0.84; spiritual activities, 0.96; and social activities, 0.93. These levels are consistent with those reported previously.[16]

Pulmonary function (FEV_1 percent of predicted) and 12-MWD were used as indicators of disease severity. Procedures for gathering these data are outlined in the next section.

The ACTRE is a structured diary for recording daily activities with the associated levels of dyspnea, fatigue, rest, and feelings of difficulty, competence, meaning, and enjoyment. Days are shown in half-hour increments and data are divided into morning, afternoon, and evening. To minimize subject burden and reduce recall difficulties, subjects are asked to complete the diary recordings 3 times each day. Subjects record their activities, the level of physical activity required (ie, asleep, mostly lying down, mostly sitting, or mostly standing or moving around), the category of activity (eg, rest, self-care, preparation, and planning), and the symptoms, meaning, and enjoyment associated with each activity. The ACTRE was developed and validated for use in patients with rheumatoid arthritis[18,19] and has been used to evaluate symptoms and activities in patients with fibromyalgia.[20] For purposes of this study, minor adjustments were made in the ACTRE symptom questions (dyspnea rather than pain). The percentage of wake time during which the subject experienced activity-related symptoms or performance difficulty was used in this study.

The Dual-Mode Mini-Motionlogger actigraph is a battery-driven wrist-worn device (accelerometer), about the size and weight (< 3 oz) of a watch, that counts and records movement. Deflection of the free end of the Actigraph's piezoelectric bilaminar bender element generates a voltage proportional to the deflection, which emits a fixed-rate, fixed-duration pulse recorded according to preprogrammed epochs. The device is programmed and data are downloaded through an interface device and personal computer. The sensitivity of the Actigraph to variations in activity has been demonstrated in laboratory settings.[21,22] The validity of the instrument as an indicator of functional performance has been shown in the correlation between movement frequency and 24-hour activity diary data in healthy young adults.[21] In this study, 2 modes of motion detection were used: zero-crossing mode (ZCM) and time-above-threshold (TAT). ZCM counts the number of times per epoch the transducer is displaced above and below the reference threshold, estimating the *frequency* of movement. TAT enables a 10 Hz counter whenever suprathreshold activity is reached, estimating *duration* of movement in number of millisecond counts stored. To stabilize within-subject movement variability, mean TAT and ZCM values were divided by their standard deviations. Thus, values associated with habitual movement received greatest weight; values associated with idiosyncratic movements received less weight.[22]

Finally, 2 instruments were selected to evaluate the concurrent validity of selected subscales of the FPI: the FSQ and the DASI. The FSQ is a brief (28-item) self-report instrument designed to measure functioning in ambulatory patients.[9,23] Items from 2 of the 6 dimensions of the FSQ were consistent with the intent of the FPI[1] and were used in the analyses. The 2 dimensions were: basic ADL (consisting of 3 items related to self-care such as bathing); and IADL (consisting of 6 items related to intermediate activities such as shopping). The FSQ has been tested for reliability and validity in various clinical populations, and has been used as an indicator of functional status in a number of studies.[24–30] Internal consistency reliability estimates (Cronbach's coefficient alpha, α) for the ADL and IADL subscales were 0.79 and 0.82, respectively, in 1,153 ambulatory patients.[9,31] Cronbach's α for these 2 subscales in the current sample was 0.81 and 0.80, respectively.

The DASI is designed to measure physical fitness and cardiac functional capacity indirectly, through a brief (12 item) self-administered questionnaire about the subject's ability to perform various activities.[8] Each activity is weighted according to its known metabolic cost in metabolic equivalent units. The Spearman correlation coefficient (r_s), describing the relationship between the DASI and peak oxygen uptake, was 0.81 in the initial instrument development sample, and 0.58 in the instrument validation sample, ($p < 0.0001$). The DASI was also correlated with the Canadian Cardiovascular Society Classification in these 2 samples ($r_s = 0.58$ and $r_s = 0.49$, respectively). The internal consistency reliability for the DASI in the present study was 0.88.

Procedures

The study was approved by the Johns Hopkins University Institutional Review Board. Subjects completed a questionnaire booklet that included the FPI, a demographic questionnaire, and additional validation measures. Each patient was instructed in the completion of the ACTRE and participated in a minimum of one practice session. The patients were instructed to wear the actigraph as they would a watch, continuously from the time they left the clinic until they returned. Although the instrument is water-resistant, they were asked to remove it during bathing to reduce the probability of mechanical failure. In order to avoid weekend effects, all monitoring took place Monday through Friday.

Subjects performed PFTs and 12-MWD tests immediately prior to or after the monitoring period. Twenty-two of the 23 subjects completed PFTs within 24 hours, and 20 completed the 12-MWD test within this time frame. One subject completed pulmonary function and 12-MWD tests within 14 days; 2 were unable to complete the 12-MWD.

PFTs were performed by an individual trained and experienced in spirometry, following standard procedures recommended by the American Thoracic Society, and according to approved standards at the Johns Hopkins Asthma and Allergy Center Pulmonary Function Laboratory.[32] The 12-MWD tests were conducted in accordance with the recommendations of McGavin et al.[33] Subjects were first oriented to the walk environment and instructed in the rating scales of exertion[34,35] and breathlessness.[36,37] A 2-minute practice walk was conducted, followed by a rest

Table 1. Sample Sociodemographic and Clinical Characteristics

Sociodemographic		Clinical	Mean ± SD
Sample size	22	Duration of illness (years)	11.32 ± 10.72
Gender	13 Men		
	9 Women	Pulmonary function	
		FEV_1 (liters)	0.99 ± 0.46
Age (years)	64.0 ± 10.10	FEV_1 predicted	37.30 ± 13.99
		FEV_1/FVC	0.47 ± 0.12
Marital status	*n* (%)		
Married	12 (55)		
Widowed	6 (27)	12-Minute walking distance test	
Divorced, separated, single	4 (18)	Distance walked (feet)	1682.23 ± 872
		Pulse elevation (beats/min)	12.85 ± .64
Median income	$10–30,000	Post walk dyspnea[34]	5.07 ± 2.90
		Perceived exertion (RPE)	12.65 ± 3.05
Education	*n* (%)		
< High school	7 (32)	Functional Status Questionnaire (FSQ)	
High school diploma	9 (41)	Activities of Daily Living (ADL)	77.27 ± 1.27
≥ High school	6 (27)	Intermediate ADL	52.17 ± 22.31
Employment status	*n* (%)	Duke Activity Status Index (DASI) (*n* = 19)	9.98 ± 12.18
Retired or disabled	16 (73)		
Full- or part-time	2 (9)	Actigraph (*n* = 20)	
Other	4 (18)	Zero crossing mode (counts/msec)	1.81 ± .35
Living arrangements	*n* (%)	Time above threshold (msec/movement)	1.77 ± .37
Living alone	5 (23)		
Living with another	17 (77)		

n = number; FEV_1 = forced expiratory volume in the first second; FVC = forced vital capacity; FEV_1/FVC = ratio of FEV_1 to FVC; RPE = rate of perceived exertion.

period to baseline. Patients then recorded their resting breathlessness and proceeded with the test. At the completion of the test, each subject rated his or her perceived exertion and repeated their rating of breathlessness. During both the practice and 12-MWD test, subjects were accompanied by a trained research nurse who provided standardized encouragement, monitored oxygen saturation and heart rate, and carried portable oxygen for the 5 subjects who required supplement.

Data Analysis

Pearson correlation procedures were used to evaluate the relationship between the FPI and each of the validation indices. Because the study was exploratory, no adjustments were made for multiple testing procedures. Results were examined in light of Cohen's criteria for interpreting the size of correlation coefficients, where an r of 0.10 is considered small, an r of 0.30 is considered medium, and an r of 0.50 is considered large.[38] According to Cohen, many coefficients reported in the behavioral sciences, and most validity coefficients (correlations between a measure and its criterion), fall into the medium effect category.

Results

Thirteen men and 10 women participated in the study; 1 woman employed as a night shift worker was excluded from the analyses because of the instability of her sleep and activity patterns. Table 1 shows sociodemographic and clinical characteristics of the participants. Most were retired and living with a spouse or adult child. Subjects felt their disease was moderate to severe, reporting a mean rating of 2.77 (± 0.69) on a scale of 1 (mild) to 4 (very severe). They also perceived that their breathing problems severely affected their activity, with a mean rating of 3.77 (± 0.92) on a scale of 1 (mild) to 4 (very severe). According to the ACTRE diary, subjects spent 34% ± 14% of their awake activity time engaged in activities such as standing or walking. While awake, subjects experienced difficulty performing activities 16% ± 21% of the time,

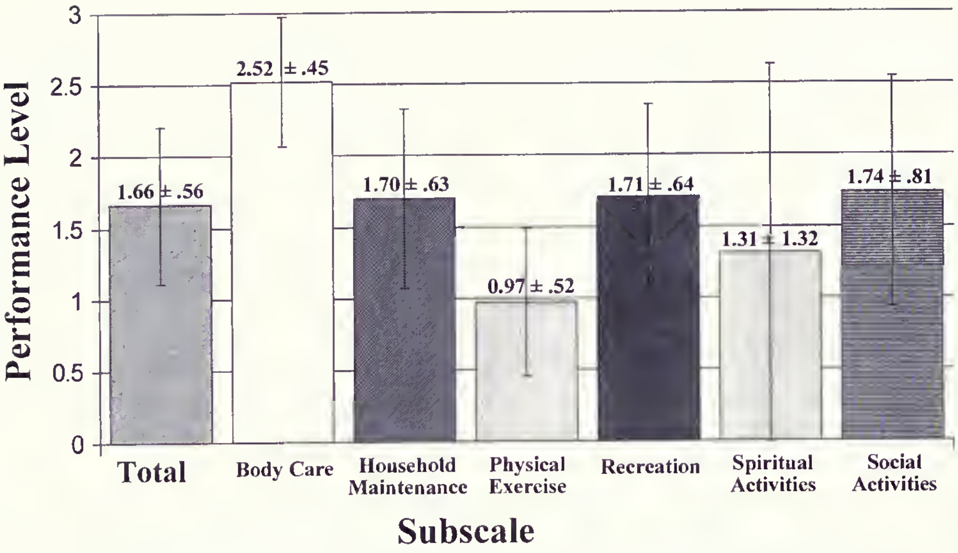


Figure 1. Functional Performance Inventory Profile. Higher scores indicate higher levels of performance. Values are mean scores ± standard deviation. Range for each scale and total score: 0–3.

Table 2. Pearson Correlation Coefficients between Functional Performance Inventory Scores and Validation Measures

Functional Performance Inventory Subscale	Validation Measure							
	Pulmonary function and exercise tolerance		NIH Activity Record Diary			Other subjective performance measures		
	FEV_1% ($n = 22$)	12-MWD ($n = 21$)	Dyspnea ($n = 22$)	Fatigue ($n = 22$)	Difficulty ($n = 22$)	FSQ-ADL ($n = 19$)	FSQ-IADL ($n = 19$)	DASI ($n = 19$)
Body Care	0.23	0.45*	−0.27	−0.25	−0.62†	0.77†	0.50†	0.01
Household maintenance	0.61†	0.64†	−0.60†	−0.63†	−0.71†	0.76†	0.81†	0.55*
Physical exercise	0.44*	0.32	−0.32	−0.42*	−0.56†	0.52*	0.50*	0.10
Recreation	0.45*	0.53*	−0.66†	−0.65†	−0.72†	0.57†	0.71†	0.54*
Spiritual activities	0.34	0.20	−0.32	−0.36	−0.24	0.11	0.23	0.14
Social activities	0.48*	0.44*	−0.56†	−0.54†	−0.73†	0.52*	0.64†	0.64†
Total score	0.55†	0.52*	−0.59†	−0.62†	−0.71†	0.61†	0.70†	0.43

FEV_1 = Forced expiratory volume in the first second. 12-MWD = 12-minute walking distance test. FSQ-ADL = Functional Status Questionnaire – Activities of Daily Living. FSQ-IADL = Functional Status Questionnaire – Intermediate Activities of Daily Living. DASI = Duke Activity Status Index.

* $p \leq 0.05$.

† $p \leq 0.01$.

dyspnea 36% ± 30% of the time, and fatigue 32% ± 24% of the time.

Figure 1 shows the FPI profile for the participants. Patients reported the poorest performance in the physical exercise domain. Body care was performed with the least difficulty. The greatest variance was found in spiritual activities. The range of scores for each of the 6 subscales were as follows: 1.44–3.00; 0.60–2.67; 0.14–2.00; 0.55–2.64; 0.00–3.00; and 0.17–3.00, respectively. The range for the FPI total score in this sample was 0.63–2.48.

Table 2 shows Pearson product moment correlations describing the relationship between the FPI and the validation measures. All of the coefficients were in the predicted direction, and ranged from 0.11 to 0.81. Statistically significant correlations were found between the FPI total score and each of the measures, with the exception of the DASI. The household maintenance, recreation, and social activities subscales correlated with all of the validation measures.

Table 3 shows the Pearson product moment correlations describing the relationship between the FPI and movement, as indicted by the ZCM and TAT counts of the actigraph. The ZCM and TAT movement indicators were significantly correlated with the household maintenance subscale of the FPI. Correlations between the body care and social activity scales and the objective movement in-

Table 3. Pearson Correlation Coefficients between Functional Performance Inventory Scores and Movement

Functional Performance Inventory Subscale	Movement Indicator	
	Zero-Crossing Mode ($n = 17$)	Time Above Threshold ($n = 17$)
Body Care	0.38	0.30
Household Maintenance	0.52*	0.53*
Physical Exercise	0.27	0.31
Recreation	0.19	0.19
Spiritual Activities	−0.15	−0.15
Social Activities	0.37	0.37
Total Score	0.26	0.25

*$p < 0.05$.

dicators were moderate, but not statistically significant due to lack of statistical power ($\beta = 0.66$).

Discussion

For a functional performance outcome measure to be consistent with and sensitive to the goal of maintaining or improving function, it should capture the domains and elements of daily activity that the patients themselves identify as important and meaningful yet difficult to perform. It should demonstrate construct and concurrent validity by performing in a manner consistent with clinical observations and theoretical expectations. The purpose of this study was to explore the extent to which the FPI correlates with clinical indicators of disease severity, daily recordings of activity-related symptoms and difficulty, movement (an objective indicator of performance), and alternative subjective indicators of activity level. Results of the study should be interpreted in light of the sample size, which was a function of the costs of a clinical study of this nature, including the labor, materials, and subject burden associated with the clinic visit, daily diary, and the actigraph monitoring and analyses. Coefficients should be examined in terms of both their magnitude and their statistical significance, with each serving as indicator of the FPI's validity and the need for further research.

Correlations between the FPI and indicators of disease severity were moderate to strong, and statistically significant. Coefficients between the FPI total score and FEV_1 percent of predicted and 12-MWD were strong, as were those involving the household maintenance, recreation, and social activities subscales. Correlations between disease severity and physical exercise, body care, and spiritual activities were moderate. The relationship between FEV_1 percent of predicted and the FPI was stronger than the previous validation study, where coefficients ranged from 0.12 (spiritual activity) to 0.36 (household maintenance and physical activity), and 0.34 with the total score.[16] The present study was designed to achieve greater precision in the estimation of pulmonary function and its relationship to the FPI by gathering PFT data in the laboratory close to the time the FPI was completed; because of the large sample size of the previous study, PFT estimations were based on chart audit. It is likely, therefore, that the correlations reported here are more accurate estimates of the FPI's validity. The fact that the coefficients were higher than previous studies of pulmonary function, walk distance, and daily activity in patients with COPD, using measures such as the SIP,[39–43] suggests the FPI is more sensitive to the effects of COPD on performance of day-to-day activities.

The modest relationship between the 12-MWD and spiritual activities was not surprising, given the relatively sedentary nature of these activities. The correlation between pulmonary function and spiritual activities was moderate but did not reach statistical significance, because of the small sample size. This subscale was included in the FPI because the patients themselves identified attendance of religious services, ceremonies, and study or discussion groups as important components of performance.[16,17] Further research is needed to determine the contribution of spiritual activity to the instrument's ability to predict and understand the effects of disease on activity.

As would be expected from clinical observations, patients who reported more activity-related dyspnea, fatigue, and difficulty in their daily diaries also reported lower levels of performance on the FPI. Correlations between the FPI and the activity diary were moderate to large, with household maintenance, recreation, and social activities being the most powerful correlates.

The relationship between objective and subjective measurement of performance has not been examined previously in COPD patients. In this study, correlations between the FPI and actigraph indicators of movement were small to moderate and not statistically significant, with the exception of household maintenance. The magnitude of the coefficients was similar to that reported by Vercoulen et al[44] for the relationship between accelerometer counts and subjective activity measures in patients with chronic fatigue syndrome. Sager et al[45] found moderate to poor agreement between self-report and performance-based assessment of basic ADLs in elderly patients hospitalized for acute or chronic conditions.

Perhaps the low correlation between objective and subjective measures in each of these studies is a function of low activity levels in these populations. Clinically, we know that many people with severe COPD conserve their energy, not unlike those with chronic fatigue and the hospitalized elderly. In fact, the mean activity counts over 72 hours of subjects in this study were considerably lower than counts associated with controlled sedentary activity in a laboratory setting (conversing, writing, reading, playing cards, estimated to be equivalent to 1 to 2 metabolic equivalent units), where mean stabilized ZCM and TAT values were 2.05 ± 0.57 and 2.51 ± 1.04, respectively.[22] For context, mean values associated with moderate activity in a laboratory setting are 3.63 ± 0.91 and 6.25 ± 3.86, while those associated with heavy activity are 3.69 ± 0.64 and 8.13 ± 4.28, respectively.[22] Diary data indicated subjects were relatively inactive, spending 65% of their waking hours sitting or lying down. Recreational activities (38% of waking hours) were generally sedentary. Self-care and household activities, though each representing a relatively small portion of the day (15% and 13% of waking hours, respectively), generally involved standing, walking, lifting, or moving. As would be expected, the relationship between the FPI and actigraph counts was moderate for the total score, weakest for the recreational subscale, and strongest for the household maintenance and body care subscales, though having the subjects remove the actigraph while bathing or showering may have attenuated the latter correlation to some degree. The fact that TAT and ZCM were significantly related to the FSQ-IADL score (0.54 and 0.48 respectively, $p < 0.05$), and to a lesser extent to the FSQ-ADL score (0.43 and 0.37 respectively, not significant) supports this interpretation. The higher correlation between movement frequency and 24-hour activity diary in active, healthy adults reported by Patterson et al[21] ($r = 0.80$; $r = 0.53$, excluding sleep time) offers further evidence that the low correlation found in this study is a function of low levels of activity.

The last validation test in this study was the extent to which the FPI was related to other, less comprehensive subjective measures of performance. The magnitude of the correlation coefficients between the FPI and the FSQ and DASI support the validity of the FPI, and suggest that the instrument is measuring unique dimensions of functional performance not measured by the other instruments. The strongest correlations were between the FPI body care and the FSQ-ADL (comprised of items related to eating, dressing, bathing, moving in and out of chairs or bed, walking indoors), between household maintenance and the FSQ-IADL (comprised of items related to climbing stairs, housework, errands, driving, and vigorous activities, including heavy lifting), and between household maintenance and the DASI (in which 6 of the 12 items address activities and work in and around the home), supporting the convergent validity of the FPI. The weakest correlations involved the spiritual activities scale, a domain not addressed by the other instruments. Correlations between the FPI total score and the FSQ and DASI indicate that only 18–49% of the variance in the FPI can be explained by these measures. The pattern and size of the correlations between the FPI, FSQ, and DASI generally correspond to those reported previously; correlations between the DASI and the body care and physical exercise subscales were substantially higher in the larger sample ($r = 0.51$ and $r = 0.59$, respectively).[16] The results are also similar to those reported by Larson et al,[46] in which the correlation between the FPI total and the Physical Scale of the SIP and the Physical Functioning subscale of the Short-Form 36 were −0.60 (36% common variance) and 0.69 (48% common variance), respectively. The extent to which the remaining variance contributes to the sensitivity of the instrument to change over time and to clinically meaningful change with treatment should be examined.

In summary, the results of this study suggest the FPI is a useful measure for evaluating subjective performance in patients with COPD, providing both a parsimonious overall score and a performance profile across 6 domains of activity that patients identified as important and that clinicians confirmed as clinically meaningful. The instrument demonstrated evidence of clinical validity through correlations with pulmonary function, walk distance, symptoms, alternative but less comprehensive subjective measures of activity and—to a certain extent—with an objective indicator of activity. Further research on responsiveness and the definition of clinically meaningful change scores for the FPI is warranted.

ACKNOWLEDGMENTS

We greatly appreciated the assistance of Kathy Fedenko RN of the NIH and Robert Wise MD and Cynthia Rand PhD of the Johns Hopkins Asthma and Allergy Center during subject recruitment and data collection.

REFERENCES

1. Leidy NK. Functional status and the forward progress of merry-go-rounds: toward a coherent analytical framework. Nurs Res 1994; 43(4):196–202.
2. Leidy NK. Using functional status to assess treatment outcomes (editorial). Chest 1994;106(6):1645–1646.
3. de Bruin AF, de Witte LP, Stevens F, Diederiks JP. Sickness Impact Profile: the state of the art of a generic functional status measure. Soc Sci Med 1992;35(8):1003–1014.
4. Larson J, Leidy NK. Chronic obstructive pulmonary disease: strategies to improve functional status. In: Fitzpatrick J, editor. Annual review of nursing research. New York: Springer; 1998:253–286.
5. Lareau SC, Breslin EH, Meek PM. Functional status instruments: outcome measure in the evaluation of patients with chronic obstructive pulmonary disease. Heart Lung 1996;25(3):212–224.
6. Leidy NK. Functional performance in people with chronic obstructive pulmonary disease. Image J Nurs Sch 1995;27(1):23–34.
7. Granger CV, Albrecht GL, Hamilton BB. Outcome of comprehensive medical rehabilitation: measurement by PULSES Profile and the Barthel Index. Arch Phys Med Rehabil 1979;60(4):145–154.
8. Hlatky MA, Boineau RE, Higginbotham MB, Lee KL, Mark DB, Califf RM, et al. A brief self-administered questionnaire to determine functional capacity (The Duke Activity Status Index). Am J Cardiol 1989;64(10):651–654.
9. Jette AM, Davies AR, Cleary PD, Calkins DR, Rubenstein LV, Fink A, et al. The Functional Status Questionnaire: reliability and validity

when used in primary care. J Gen Intern Med 1986;1(3):143–149. *Published erratum appears in J Gen Intern Med 1986;1(6):427.*

10. Katz S. Assessing self-maintenance: activities of daily living, mobility, and instrumental activities of daily living. J Am Geriatr Soc 1983;31(12):721–727.
11. Mahoney RI, Barthel DW. Functional evaluation: The Barthel Index. Md State Med J 1965;14:61–65.
12. Hutchinson TA, Boyd NF, Feinstein AR, Gonda A, Hollomby D, Rowat B. Scientific problems in clinical scales, as demonstrated in the Karnofsky Index of Performance Status. J Chronic Dis 1979; 32(9–10):661–666.
13. Lareau SC, Carrieri-Kohlman V, Janson-Bjerklie S, Roos PJ. Development and testing of the Pulmonary Functional Status and Dyspnea Questionnaire (PFSDQ). Heart Lung 1994;23(3):242–250.
14. Lareau SC, Meek PM, Roos PJ. Development and testing of the modified version of the Pulmonary Functional Status and Dyspnea Questionnaire (PFSDQ-M). Heart Lung 1998;27(3):159–168.
15. Weaver TE, Narsavage GL, Guilfoyle MJ. The development and psychometric evaluation of the Pulmonary Functional Status Scale: an instrument to assess functional status in pulmonary disease. J Cardiopulm Rehabil 1998;18(2):105–111.
16. Leidy NK. Psychometric properties of the Functional Performance Inventory in patients with chronic obstructive pulmonary disease. Nurs Res 1999;48(1):20–28.
17. Leidy NK, Haase JE. Functional performance in people with chronic obstructive pulmonary disease: a qualitative analysis. Adv Nurs Sci 1996;18(3):77–89.
18. Gerber LH, Furst GP. Validation of the NIH activity record: a quantitative measure of life activities. Arthritis Care Res 1992;5(2):81–86.
19. Gerber LH, Furst GP. Scoring methods and application of the activity record (ACTRE) for patients with musculoskeletal disorders. Arthritis Care Res 1992;5(3):151–156.
20. Henriksson C, Gundmark I, Bengtsson A, Ek AC. Living with fibromyalgia: consequences for everyday life. Clin J Pain 1992;8(2): 138–144.
21. Patterson SM, Krantz DS, Montgomery LC, Deuster PA, Hedges SM, Nebel LE. Automated physical activity monitoring: validation and comparison with physiological and self-report measures. Psychophysiology 1993;30(3):296–305.
22. Leidy NK, Abbott RD, Fedenko KM. Sensitivity and reproducibility of the dual-mode actigraph under controlled levels of activity intensity. Nurs Res 1997;46(1):5–11.
23. Yarnold PR, Bryant FB, Repasy AB, Martin GJ. The factor structure and cross-sectional distributional properties of the Beth Israel/UCLA Functional Status Questionnaire. J Behav Med 1991;14(2):141–153.
24. Allen JK, Becker DM, Swank RT. Factors related to functional status after coronary artery bypass surgery. Heart Lung 1990;19(4):337–343.
25. Barsky AJ, Cleary PD, Klerman GL. Determinants of perceived health status of medical outpatients. Soc Sci Med 1992;34(10):1147–1154.
26. Einarsson G, Grimby G. Disability and handicap in late poliomyelitis. Scand J Rehabil Med 1990;22(2):113–121.
27. Katz JN, Larson MG, Phillips CB, Fassel AH, Liang MH. Comparative measurement sensitivity of short and longer health status instruments. Med Care 1992;30(10):917–925.
28. Reuben DB, Rubenstein LV, Hirsch SH, Hays RD. Value of functional status as a predictor of mortality: results of a prospective study. Am J Med 1992;93(6):663–669.
29. Rubenstein LV, Calkins DR, Young RT, Cleary PD, Fink A, Kosecoff J, et al. Improving patient function: a randomized trail of functional disability screening. Ann Intern Med 1989;111(10):836–842.
30. Tedesco C, Manning S, Lindsay R, Alexander C, Owen R, Smucker ML. Functional assessment of elderly patients after percutaneous aortic balloon valvuloplasty: New York Heart Association classification versus functional status questionnaire. Heart Lung 1990;19(2): 118–125.
31. Jette AM, Cleary PD. Functional disability assessment. Phys Ther 1987;67(12):1854–1859.
32. American Thoracic Society. Lung function testing: selection of reference values and interpretive strategies. Am Rev Respir Dis 1991; 144(5):1202–1218.
33. McGavin CR, Gupta SP, Lloyd EL, McHardy GJ. Physical rehabilitation for the chronic bronchitic: results of a controlled trail of exercises in the home. Thorax 1977;32(3):307–311.
34. Borg GA. Psychophysical bases of perceived exertion. Med Sci Sports Exerc 1982;14(5):377–381.
35. Dunbar CC. Practical use of ratings of perceived exertion in a clinical setting. Sports Med 1993;16(4):221–224.
36. Burdon JG, Juniper EF, Killian KJ, Hargreave FE, Campbell EJ. The perception of breathlessness in asthma. Am Rev Respir Dis 1982; 126(5):825–828.
37. Wilson RC, Jones PW. A comparison of the visual analogue scale and modified Borg scale for the measurement of dyspnoea during exercise. Clin Sci 1989;76(3):277–282.
38. Cohen J. Statistical power analysis for the behavioral sciences, 2nd ed. Hillsdale NJ: Lawrence Erlbaum; 1988.
39. Jones PW, Baveystock CM, Littlejohns P. Relationships between general health measured with the Sickness Impact Profile and respiratory symptoms, physiological measures, and mood in patients with chronic airflow limitation. Am Rev Respir Dis 1989;140(6):1538–1543.
40. McSweeny AJ, Grant I, Heaton RK, Adams KM, Timms RM. Life quality of patients with chronic obstructive pulmonary disease. Arch Intern Med 1982;142(3):473–478.
41. Nocturnal Oxygen Therapy Trial (NOTT) Group. Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive lung disease: a clinical trial. Ann Intern Med 1980;93(3):391–398.
42. Prigatano GP, Wright EC, Levin D. Quality of life and its predictors in patients with mild hypoxemia and chronic obstructive pulmonary disease. Arch Intern Med 1984;144(8):1613–1619.
43. Schrier AC, Dekker FW, Kaptein AA, Dijkman JH. Quality of life in elderly patients with chronic nonspecific lung disease seen in family practice. Chest 1990;98(4):894–899.
44. Vercoulen JH, Bazelmans E, Swanink CM, Fennis JF, Galama JM, Jongen PJ, et al. Physical activity in chronic fatigue syndrome: assessment and its role in fatigue. J Psychiatr Res 1997;31(6):661–673.
45. Sager MA, Dunham NC, Schwantes A, Mecum L, Halverson K, Harlowe D. Measurement of activities of daily living in hospitalized elderly: a comparison of self-report and performance-based methods. J Am Geriatr Soc 1992;40(5):457–462.
46. Larson JL, Kapella MC, Wirtz S, Covey MK, Berry J. Reliability and validity of the Functional Performance Inventory in patients with moderate to severe chronic obstructive pulmonary disease. J Nurs Meas 1998;6(1):55–73.

Dry Powder Inhalers

Rajiv Dhand MD and James Fink RRT

Introduction
Aerosol Generation by Dry Powder Inhalers
Factors Influencing Aerosol Delivery
  Device Design
  Particle Characteristics
  Environmental Conditions
  Inspiratory Flow
  Breath-Hold
  Exhalation into the Device
  Patient-Related Factors
Aerosol Deposition in the Lung
  Particle Size Distribution In Vitro vs In Vivo
  Deposition of Radiolabeled Aerosol In Vivo
  Plasma Levels after Inhalation
  Clinical Efficacy Studies
Drugs Available as Inhalable Dry Powders
Problems with Use of Dry Powder Inhalers
Comparison of Pressurized Metered Dose Inhalers and Dry Powder Inhalers
Final Recommendations

[Respir Care 1999;44(8):940–951] *Key words: dry powder inhaler, metered dose inhaler, inhalation device, aerosol, inhaled medication, micronized powder.*

Introduction

Pressurized metered-dose inhalers (pMDIs) are the most popular devices for administering inhaled drugs. However, more than 80 countries have signed the Montreal Protocol to ban the production of chlorofluorocarbons in the next few years, which necessitates the replacement of chlorofluorocarbon-propelled pMDIs with other aerosol delivery devices. Among the developing alternative means of delivering therapeutic aerosols, dry powder inhalers (DPIs) are gaining in popularity. Crystalline or powder drug inhalation is inexpensive, does not depend on the use of chlorofluorocarbons, and does not require coordination between inhalation and device actuation. Also, recent improvements in the design, ease of use, and multidose capability make DPIs attractive alternatives to pMDIs for aerosol therapy in ambulatory patients.

Aerosol Generation by Dry Powder Inhalers

DPIs create aerosols by drawing air through an aliquot of dry powder. The powder contains either micronized (< 5 μm in diameter) drug particles bound into loose aggregates, or micronized drug particles that are loosely bound to large (> 30 μm in diameter) lactose or glucose particles.[1] Micronized particles adhere to each other and to most surfaces, and the addition of the larger carrier particles decreases interparticulate forces in the micronized drug powder so that separation into individual respirable particles (deaggregation) occurs more readily. Thus, the carrier

---

Rajiv Dhand MD and James Fink RRT are affiliated with the Division of Pulmonary and Critical Care Medicine, Loyola University of Chicago Stritch School of Medicine, Hines VA Hospital, Hines, Illinois.

Correspondence: Rajiv Dhand MD, Division of Pulmonary and Critical Care Medicine, 111N, Hines VA Hospital, 5th and Roosevelt Road, Hines IL 60141.

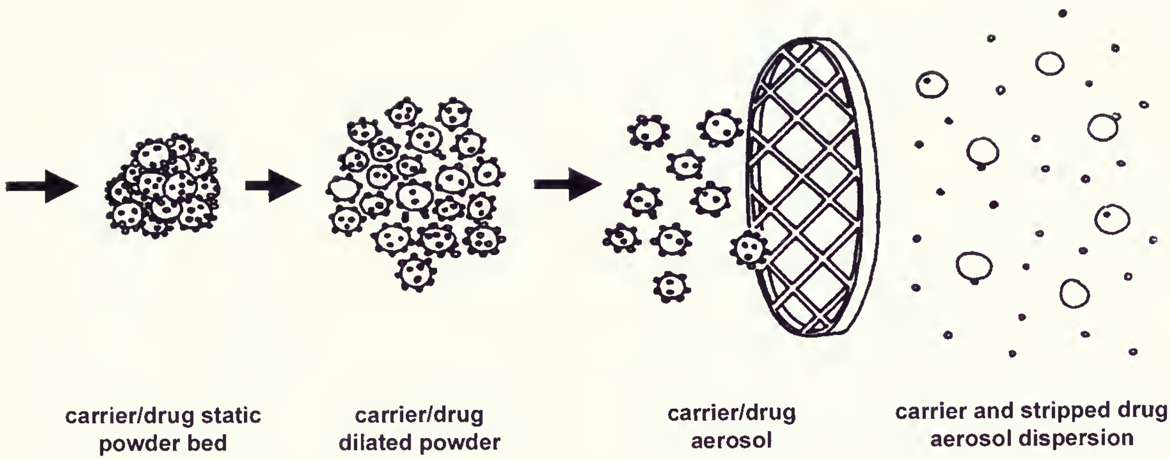

carrier/drug static powder bed
carrier/drug dilated powder
carrier/drug aerosol
carrier and stripped drug aerosol dispersion

Fig. 1. Aerosolization of dry powder. (Modified from Reference 50.)

particles aid the flow of the drug powder from the device ("flow aid"). Moreover, carriers act as "fillers" by adding bulk to the powder when the drug unit dose is very small. Usually, the drug particles are bound to carrier particles[2] and are stripped from the carrier particles by the energy provided by the patient's inhalation (Fig. 1). The release of respirable particles of the drug requires inspiration at relatively high flow rates (30–120 L/min).[3–5] The aerosol produced by a DPI contains drug particles, which are 1–2 μm in diameter, and carrier particles, which are > 30 μm in diameter. Fast inspiration results in high pharyngeal impaction of the carrier particles, and this gives the patient the sensation of having inhaled a dose.

Factors Influencing Aerosol Delivery

Table 1 lists the factors that influence drug delivery from DPIs. Some of these factors are discussed below.

Table 1. Factors Influencing Aerosol Deposition

- Device design
  - Materials
  - Resistance to flow
  - Bolus or continuous dispersion
- Particle-related
  - Size
  - Shape
  - Density
  - Roughness
  - Electrical charge
  - Presence of carrier
- Environmental
  - Humidity
  - Temperature
- Ventilatory factors
  - Tidal volume
  - Inspiratory flow rate
  - Breath-hold
- Patient-related
  - Age
  - Airway diameter
  - Type of disease
  - Dexterity

Device Design

Aerosol delivery from DPIs is influenced by the materials used in their manufacture, resistance to airflow, proper assembly of the components, and by accumulation of the powder within the device. The materials used to manufacture DPIs interact with the electrostatic charge on the particles in the powder and influence particle deposition. Moreover, the internal geometry of the device, particularly the dimensions of the inhalation channels, influences the resistance to inspiration and the inspiratory flow required to produce an aerosol. Thus, devices with higher resistance require a higher inspiratory flow to produce a dose. Inhalation through high resistance DPIs may improve drug delivery to the lower respiratory tract compared to devices with lower resistance.[6,7] For DPIs with multiple components, correct assembly of the apparatus and/or priming of the device is important for ensuring aerosolization of the dry powder. Accumulation of dry powder within DPIs alters the properties of the aerosol produced, so periodic brushing is necessary to remove residual powder.

Particle Characteristics

The fraction of the dose contained in respirable particles (diameter 1–5 μm) is the best estimate of the dose that is likely to reach the lungs.[8] DPIs produce aerosols in which most of the drug particles are in the respirable range. However, depending on the drug formulation and device de-

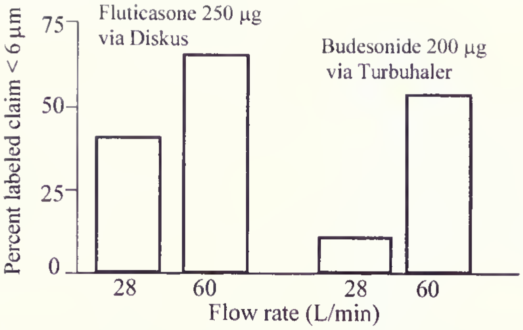


Fig. 2. Effect of inspiratory flow on fine particle mass (aerodynamic diameter < 6 μm) in aerosols produced by Diskus (fluticasone) and Turbuhaler (budesonide) dry powder inhalers. (Modified from Reference 18.)

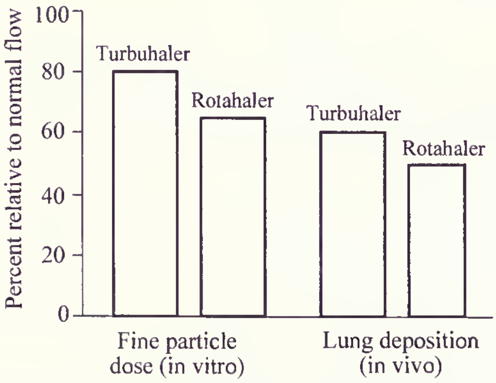


Fig. 3. Effect of inspiratory flow on fine particle mass and in vivo pulmonary deposition with the Turbuhaler and with the Rotahaler. (Modified from Reference 19.)

sign, the distribution of particle sizes within the aerosol can differ significantly among DPIs.[9]

Environmental Conditions

High ambient humidity produces clumping of the dry powder, creating larger particles that are not as effectively aerosolized and, therefore, less likely to reach the lung.[10] The powder in some DPIs is packaged within hard gelatin capsules, and high ambient humidity can soften the walls of the capsules so that they do not puncture or break cleanly, thereby reducing dispersion of the powder into the air stream. Most devices that contain drug powder in a reservoir, rather than individually sealed doses, incorporate a desiccant to prevent partial solubilization and agglomeration of the drug particles by ambient humidity. The presence of the desiccant reduces, but does not eliminate, the effects of humidity on the generation of a dry powder aerosol. The Diskhaler (Glaxo Wellcome Inc, Research Triangle Park, North Carolina) and Diskus (Glaxo Wellcome Inc, Research Triangle Park, North Carolina) are probably less susceptible to the effects of ambient humidity than other DPIs, since the dose remains sealed until the patient is ready to inhale.

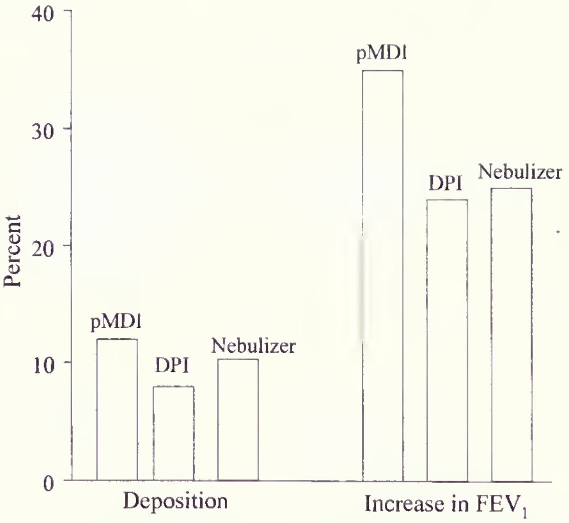


Fig. 4. Pulmonary deposition of equivalent doses of radiolabeled aerosol generated by pressurized metered dose inhaler (pMDI), dry powder inhaler (DPI), and nebulizer, and corresponding bronchodilator effects. (Modified from Reference 41.)

Inspiratory Flow

Since the energy from the patient's inspiratory flow disperses the powdered drug, the magnitude and duration of the patient's inspiratory effort influence DPI aerosol generation.[11,12] Failure to inhale at a fast enough inspiratory flow reduces the dose delivered by the DPI,[13] and increases the distribution of particle sizes within the aerosol.[14] DPI models vary in the degree to which inspiratory flow influences the dose delivered. For example, the Diskus delivered approximately 90% of the labeled dose at inspiratory flow rates of 30–90 L/min, whereas the dose delivered by the Turbuhaler (Astra US Inc, Westborough, Massachusetts) was significantly lower at an inspiratory flow of 30 L/min compared to the dose delivered at 90 L/min, and variability between doses at various inspiratory flow rates was higher with the Turbuhaler.[9,15] Other devices not yet approved for clinical use employ either a small motor and impeller or compressed gas propulsion to disperse the powder, and aerosol production by these devices is less influenced by inspiratory flow.

Breath-Hold

Breath-holding after inhalation allows greater time for drug particles to deposit by sedimentation in the airways. However, in asthmatic children, breath-holding after DPI administration of a bronchodilator did not influence the response to the medication.[16]

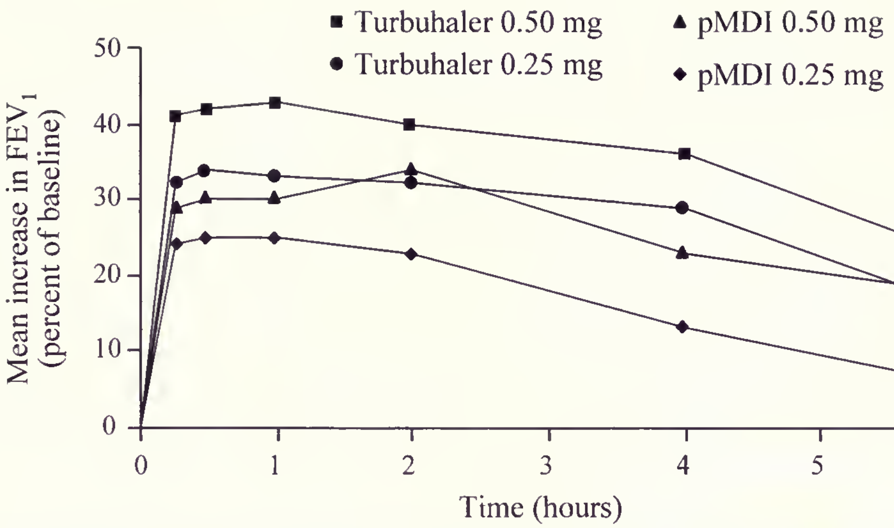


Fig. 5. Mean increase in forced expiratory volume in the first second (FEV_1) expressed as percentage increase from baseline for 6 hours after administration of terbutaline via Turbuhaler dry powder inhaler or via pressurized metered dose inhaler (pMDI). (Modified from Reference 25.)

Exhalation into the Device

Exhalation into a DPI blows the powder out of the device and reduces drug delivery. Moreover, the humidity in the exhaled air can influence subsequent aerosol generation from the DPI. Therefore, the patient must be instructed not to exhale into the DPI.

Patient-Related Factors

Because DPIs depend on inspiratory airflow to generate the aerosol, they are more susceptible to variations in age, sex, and the user's disease status than are pMDIs or nebulizers. Very young children (< 6 years old), elderly, infirm, semi-alert individuals, and those with altered mental status cannot use DPIs effectively.

Aerosol Deposition in the Lung

The deposition of drugs in the lung is assessed by (1) in vitro measurement of the particle size distribution of the aerosol, (2) in vivo measurement of the deposition of radiolabeled aerosol, (3) plasma concentrations of the drug

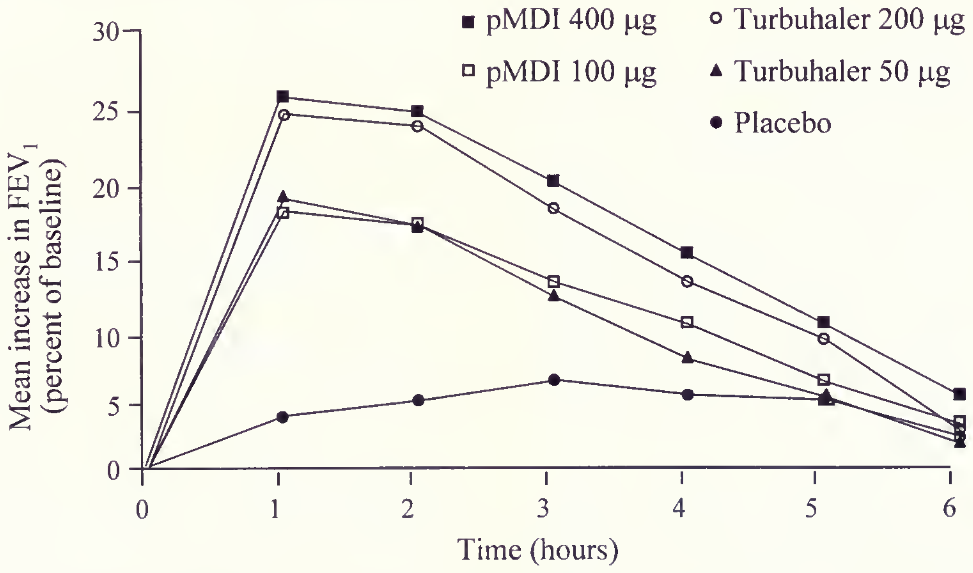


Fig. 6. Mean increase in FEV_1 expressed as percentage increase from baseline for 6 hours after administration of albuterol or placebo via Turbuhaler or via pressurized metered dose inhaler (pMDI). (Modified from Reference 27.)

Table 2. Drugs Available in the United States as Inhalable Powders

Drug	Device	Number of doses	Unit dose	Recommended dose
Albuterol sulfate	Rotahaler	1	200 μg	1–2 caps QID
Salmeterol xinafoate	Diskus	60	50 μg	1 dose BID
Budesonide	Turbuhaler	200	200 μg	1–2 doses BID
Fluticasone propionate	Diskhaler	4	50, 100, 250 μg	1–2 doses BID

BID = twice a day; QID = four times a day.

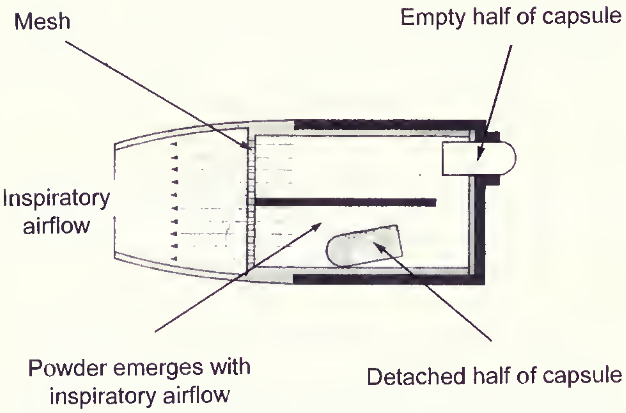


Fig. 7. Cross section of the Rotahaler (Glaxo Wellcome Inc, Research Triangle Park, North Carolina). The capsule is inserted into a port and broken into 2 pieces by twisting the inhaler. The drug is contained in the body of the capsule, which falls into the device, and the cap is retained in the port. The body of the capsule vibrates erratically when the patient inhales, dislodging the drug particles into the air stream. A mesh facilitates deaggregation of the powder. (From Reference 51, with permission.)

after inhalation, and/or (4) the response to drug administration.

Particle Size Distribution In Vitro vs In Vivo

With models that simulate clinical DPI use, the distribution of aerosol particle sizes is an indicator of the efficiency of the device. For example, in vitro investigations found $\sim$50% lower delivery of albuterol from a Rotahaler (Glaxo Wellcome Inc, Research Triangle Park, North Carolina) than from a pMDI.[17] Other investigators have found that the fine particle mass ($< 6\ \mu m$ in size) of aerosol produced by a DPI is influenced by the inspiratory flow. Although the fine particle mass does not equal the amount of aerosol deposition in the lung, it provides an estimate of the fraction of the dose that is likely to reach the lungs. At an inspiratory flow of 60 L/min, the Turbuhaler produced a significantly lower fine particle mass (approximately 50%) than the Diskus (approximately 70%).[18] Moreover, when the inspiratory flow was reduced to 28 L/min the decrease in fine particle mass was more pronounced with the Turbuhaler than with the Diskus (Fig. 2). At an inspiratory flow of 28 L/min, only $\sim$10% of the aerosol produced by the Turbuhaler contained particles $< 6\ \mu m$, compared to $\sim$30% with the Diskus. Other investigators have also found that the in vitro performance of DPIs significantly depends on inspiratory flow and inhaled volume. Hill and Slater[9] found that the fine particle mass produced by the Diskus decreased from 21% to 16% when the inspiratory flow was reduced from 60 L/min to 28.3 L/min. The corresponding values with the Turbuhaler were 18% and 6%, respectively. Thus, DPIs show significant differences in their performance in vitro.

Olson et al compared the in vitro and in vivo performance of albuterol DPIs and an albuterol pMDI in the same study.[19] The devices were tested at a "normal" inspiratory flow, then at half the normal inspiratory flow (Fig. 3). At half the normal flow, the fine particle mass and the pulmonary deposition of aerosol were reduced with both Turbuhaler and Rotahaler.

Deposition of Radiolabeled Aerosol In Vivo

Zainudin et al used albuterol mixed with technetium-99^{m}-labeled Teflon particles to compare pulmonary deposition and bronchodilator effect from equivalent doses administered by DPI or via pMDI.[20] The pMDI delivered

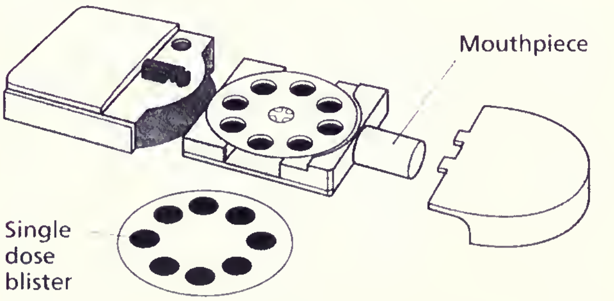
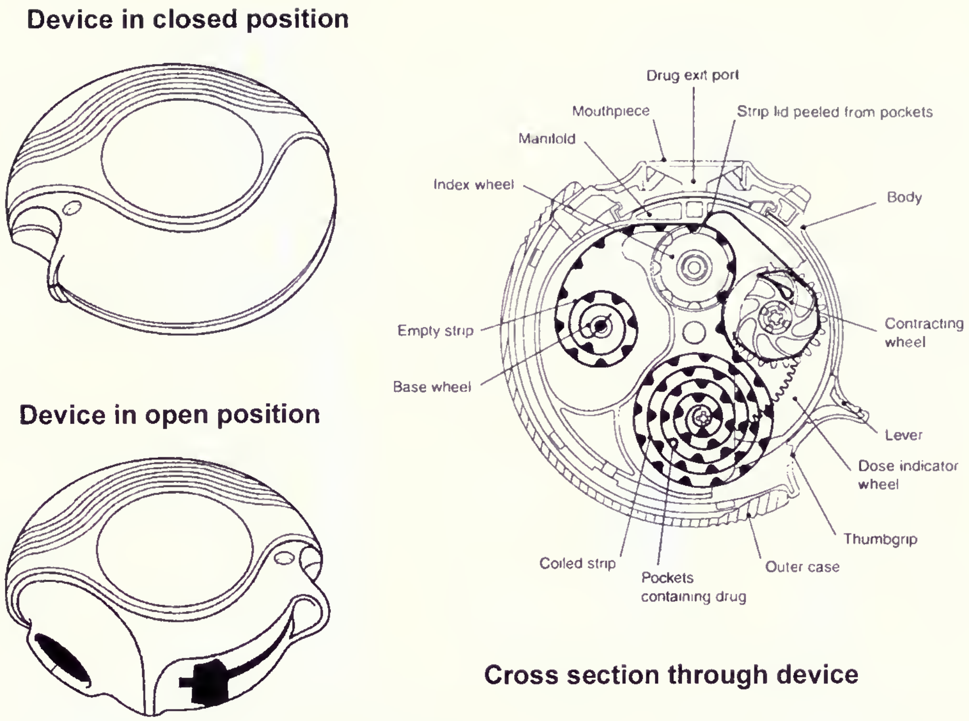


Fig. 8. The components of the Diskhaler (Glaxo Wellcome Inc, Research Triangle Park, North Carolina) multi-unit dose dry powder inhaler. The Diskhaler contains 4 or 8 numbered blisters arranged on a circular disc. Each blister contains a unit dose of drug. A needle pierces the blister when the device is primed. The number on the blister indicates the number of doses remaining in the device. (From Reference 52, with permission.)



Device in closed position

Device in open position

Cross section through device

Fig. 9. The Diskus dry powder inhaler (Glaxo Wellcome Inc, Research Triangle Park, North Carolina). The doses are contained in 60 sealed pockets along an aluminum-foil strip that is advanced by the lever. As the drug pocket reaches the mouthpiece, its cover is peeled away, making the drug available for inhalation. A dose counter indicates the number of doses remaining in the device. (Modified from Reference 52, with permission.)

a significantly greater dose to the lung (11.2 ± SE 0.8%) than the DPI (9.1 ± 0.6%), and the mean increase in the forced expiratory volume in the first second (FEV_1) with the pMDI (35.6%) was greater than that from the Rotahaler (25.2%) (Fig. 4). However, in a separate study, the Turbuhaler achieved a higher pulmonary deposition (mean 21.4%) than the pMDI (mean 10–14%).[21] Pulmonary deposition of radiolabeled aerosols has not been reported for the Diskhaler or Diskus.

Plasma Levels after Inhalation

In the "charcoal block" technique, orally administered charcoal blocks gastrointestinal absorption of the drug. With this technique, the plasma and urine levels after inhalation of the drug indicate its pulmonary absorption, provided the lung does not metabolize the drug. Using the charcoal block technique, Thorsson et al found that 32% of the nominal dose of budesonide administered with a Turbuhaler deposited in the lungs of healthy subjects, compared to 15% of the nominal dose from a pMDI.[6] In contrast, the systemic bioavailability attributed to pulmonary deposition was 13.5% for fluticasone propionate administered with a Diskhaler, versus 30% of that administered with a pMDI.[22] The charcoal block technique has not yet been used to assess pulmonary deposition with the Diskus. To summarize, the fraction of drug that successfully deposits in the lung differs between DPIs and pMDIs, and varies considerably among DPIs.

Clinical Efficacy Studies

Evaluation of therapeutic equivalence is the preferred method for comparing the performance of various devices. For bronchodilators, shallow dose-response curves can obscure the comparisons between single doses of the drug, because the response plateaus rapidly after the administration of increasing doses of the drug. Therefore, comparisons of the response to a drug given by different devices should include subtherapeutic doses of the drug. In addition, it is difficult to compare the efficacy of inhaled corticosteroids administered with different devices, and doing so requires long-term studies in a variety of patients.

Only a few investigators have compared single dose response to bronchodilators administered via DPI versus via pMDI. The response to albuterol administration via Rotahaler was similar to that obtained with half the dose administered via pMDI.[23] Similarly, 400 μg of albuterol administered via Rotahaler achieved an increase in FEV_1 comparable to that observed after 180 μg of the drug administered via pMDI.[24] In a randomized, double-blind, double-dummy study involving 13 adult patients with sta-

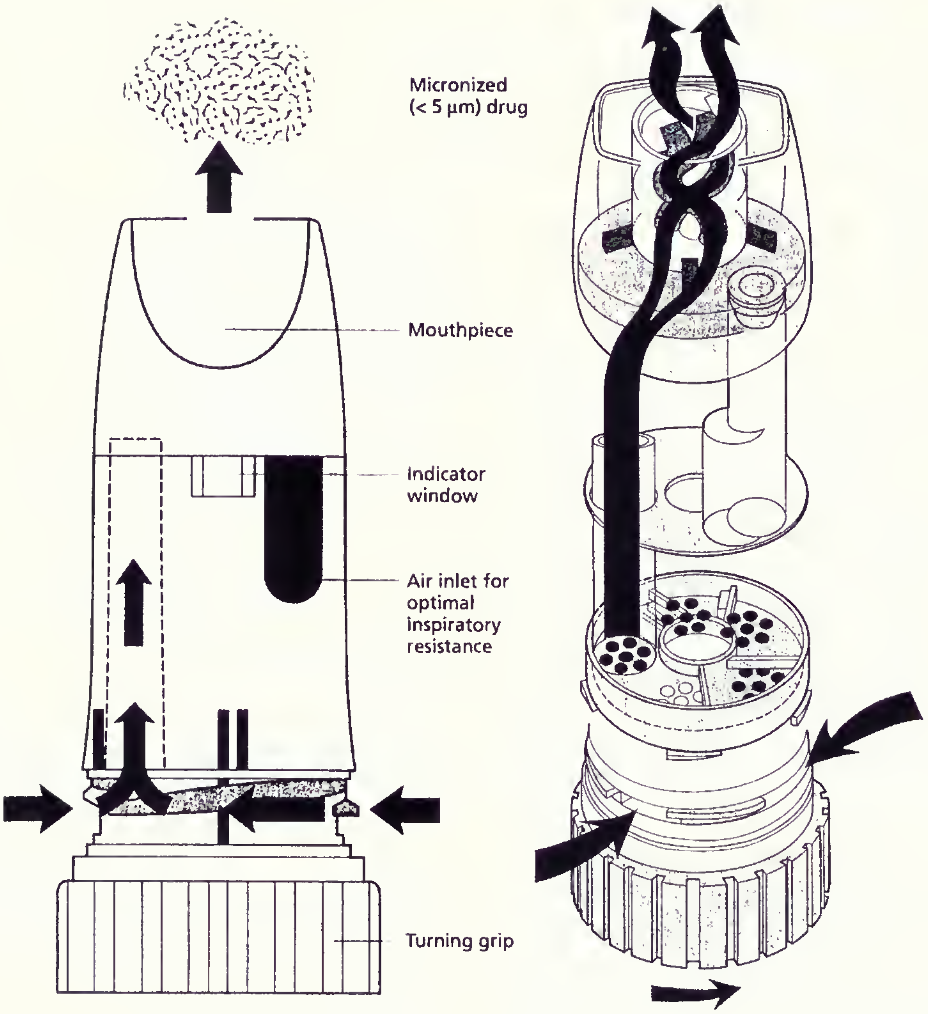


Fig. 10. Components and airflow of the Turbuhaler (Astra US Inc, Westborough, Massachusetts). The inhaler contains up to 200 doses in its reservoir. A rotating disc below the reservoir has a series of clusters of conical cavities. Turning the grip at the bottom of the device rotates the disc, and plastic scrapers above the dosing unit load the dose into a group of conical holes. The inhaler must be held upright during this operation so that a reproducible quantity of drug is fed, by gravity, into the conical cavities. A desiccant in the device protects the drug from moisture. The powder used in this device consists of micronized drug particles, without carrier particles. Since a minute amount of powder is inhaled, the patient may not experience any sensation of receiving a dose. The Turbuhaler does not have a dose counter, but a red sign appears in the dose indicator window when 20 doses remain in the device. When the patient inhales, air enters through channels at the base of the operating unit, passes through the pressure plate, through the cavities in the dosing unit, and into the inhalation channel. Turbulent air flow in the spiral channels deaggregates the particles. The twisting channels cause resistance to inhalation, so inspiratory air flow > 30 L/min is needed to obtain an adequate dose. Particles that are poorly entrained are retained in the channels and become reentrained as smaller particles during subsequent inhalations. (Modified from Reference 52, with permission.)

ble asthma, Borgstrom et al compared the deposition and pulmonary effects of single doses of terbutaline, 0.25 mg and 0.5 mg, administered via pMDI versus via Turbuhaler.[25] The total pulmonary deposition was higher with the Turbuhaler (~20%) than with the pMDI (~8%). Figure 5 shows Borgstrom et al's measurements of changes in FEV_1 from administration of terbutaline via Turbuhaler versus via pMDI. The figure suggests that administration via the Turbuhaler was more effective for both of the studied dose levels (0.25 mg and 0.5 mg), but the administration technique employed for the 2 devices may have influenced the results. Specifically, the inspiratory flow used for the Turbuhaler (60 L/min) was optimal, but the inspiratory flow used with the pMDI (~90 L/min) was 2–3-fold higher than the recommended flow for the pMDI. High inspiratory flow with a pMDI decreases the response to bronchodilator aerosols[26] and may have reduced the bronchodilation achieved by terbutaline in the Borgstrom et al study.

Another randomized, placebo-controlled, crossover study involving stable asthma patients compared the efficacy of albuterol administered via pMDI (in doses of 100

Table 3. Instructions for Use of 4 Models of Dry Powder Inhaler

Rotahaler	Diskhaler	Diskus	Turbuhaler
1. Insert capsule. 2. Twist device to break capsule. 3. Keep device level while inhaling dose. 4. Breath-hold. 5. Remove device from mouth and exhale outside device. 6. Store device in cool, dry place.	1. Remove mouthpiece cover. 2. Pull tray out from device. 3. Place disk on wheel (numbers up). 4. Rotate disk by sliding tray out and in. 5. Lift back of lid until fully upright so that needle pierces both sides of blister. 6. Keep device level while inhaling dose. 7. Breath-hold. 8. Remove device from mouth and exhale outside device. 9. Once every week, brush off any powder remaining within device. 10. Store device in cool, dry place.	1. Open the device. 2. Slide the lever. 3. Keep device level while inhaling dose. 4. Breath-hold. 5. Remove device from mouth and exhale outside device. 6. Store device in cool, dry place.	1. Twist and remove cover. 2. Hold inhaler upright (mouthpiece up). 3. Turn grip right then left until it clicks. 4. Inhale dose. Inhaler may be held upright or horizontal for this step. 5. Breath-hold. 6. Remove device from mouth and exhale outside device. 7. Replace cover and twist to close. 8. Store device in cool, dry place.

μg and 400 μg) versus via Turbuhaler (in doses of 50 μg and 200 μg).[27] Optimal inhalation technique was used for each device. With all 4 active treatments, the increase in FEV_1 was greater than with placebo (Fig. 6). The areas under the curve for increase in FEV_1 were significantly greater for the higher doses than for the lower doses, with both the pMDI and the Turbuhaler. However, the responses to the 2 higher doses and the 2 lower doses were similar between devices. This study illustrates how lower-than-recommended doses can be used to determine equivalence of therapeutic effects. The authors concluded from this study that the bronchodilator effect of albuterol administered via Turbuhaler was double that of the same dose administered via pMDI.[27]

Boulet et al compared the clinical effects of salmeterol administered via Diskus versus via Diskhaler. The increase in peak expiratory flow (PEF) after administration of salmeterol 50 μg twice daily via Diskus was comparable to that achieved with the same dose via Diskhaler, and significantly greater than that observed with placebo.[28]

Table 4. Differences in Inhalation Technique: Pressurized Metered Dose Inhaler (MDI) vs Dry Powder Inhaler (DPI)

	MDI	DPI
Need to shake inhaler	Yes	No
Actuation with inspiration	No	Yes
Inspiration	Slow, deep	Fast, prolonged
Interval between doses	30–60 s	20–30 s
Use of spacers	Yes	No
Exhalation	Not a factor	Not into device

Drugs Available as Inhalable Dry Powders

Table 2 lists the drugs currently available in the United States in inhalable dry powder formulations. The Rotahaler (Fig. 7) uses individual doses administered in gelatin capsules, which are punctured immediately before inhalation by twisting the inhaler. The Diskhaler (Fig. 8) and Diskus (Fig. 9) use individual blister packets of drug. The Turbu-

Table 5. Comparison of Performance and Convenience Factors for Pressurized Metered Dose Inhalers and Dry Powder Inhalers

	Metered Dose Inhaler	Dry Powder Inhaler
Majority of aerosol particles < 5 μm in size	Yes	Yes
High pulmonary deposition	No	No
Low mouth deposition	No	No
Reliability of dose	Yes	Conditional
Less influenced by humidity	Yes	No
Physical and chemical stability	Yes	Yes
Environmentally safe	No	Yes
Breath actuated	No	Yes
No risk of contamination	Yes	Yes
Lightweight, compact	Yes	Yes
Multiple doses	Yes	Yes
Dose indicator	No	Yes
Dose counter	No	Yes
Inexpensive	Yes	Yes
Reusable	No	No
Easy and quick operation	Conditional	Conditional
Suitable for all ages	No	No
Suitable for multiple clinical situations	Conditional	No

Table 6. Long-Term Effects of Asthma Drugs: Pressurized Metered Dose Inhaler (MDI) vs Dry Powder Inhaler (DPI)

Drug	Study design/reference	*n*	Dose via MDI	Dose via DPI (model)	Remarks
Albuterol	Double blind, crossover, 24 weeks[43]	38	200 μg	200 μg (Rotahaler)	• Devices exhibited comparable control of symptoms • Increased frequency of using DPI during third month of treatment
Albuterol	Open, crossover, 6 weeks[44]	14	200μg	400 μg (Rotahaler)	• Trend towards higher mean increase in morning and evening PEF rate with DPI
Albuterol	Double-blind, randomized, double-dummy, parallel, 12 weeks[45]	231	180 μg	200 μg (Rotahaler)	• DPI and MDI equally effective
Salmeterol	Randomized, double-blind, double-dummy, parallel, 12 weeks‡	240	50 μg BID	50 μg BID (Diskus)	• Significant improvement in pulmonary function with both devices vs placebo • No significant difference between devices
Salmeterol*	Randomized, double-blind, double-dummy, parallel, 12 weeks‡	258	50 μg BID	50 μg BID (Diskus)	• Significant improvement in pulmonary function with both devices vs placebo • Greater bronchodilator response with MDI vs Diskus at baseline
Fluticasone	Double-blind, randomized, parallel, 6 weeks[46]	585	500 μg/day or 1000 μg/day BDP	500 μg/day (Diskhaler)†	• No difference in outcome between 2 formulations of Fluticasone
Fluticasone	Double-blind, double-dummy randomized 52 weeks‡	381	500 μg/BID	500 μg/BID (Diskhaler)	• All patients showed improvement in outcome • No difference in efficacy between devices
Budesonide	Open, randomized, cross-over, 8 weeks[47]	28	400/800 μg BID	400/800 μg BID (Turbuhaler)	• Morning PEF higher with Turbuhaler
Budesonide	Open, randomized, cross-over, 8 weeks[48]	24	400 μg BID	400 μg BID (Turbuhaler)	• No difference between devices
Budesonide	Open, randomized, parallel, 52 ± 4 weeks[29]	1,004	Bronchodilator and inhaled corticosteroid	Terbutaline and Budesonide (Turbuhaler)	• Decrease in asthma exacerbations, physician contacts, and symptoms with DPI
Budesonide	Open, randomized, parallel, 8 weeks[49]	58	0.4, 0.8, 1.6, 2.4 mg/day	0.4, 0.8, 1.6, 2.4 mg/day (Turbuhaler)	• Dose-dependent improvement • No difference between devices

---

* The response to salmeterol delivered by Diskus might not be clinically equivalent to that with salmeterol delivered by MDI in all patients.

† The MDI produces aerosol with a higher fine particle fraction (35% vs 12% of nominal dose), and achieves higher pulmonary deposition (30% vs 13.5% of nominal dose) than the DPI.

‡ Glaxo Wellcome Inc, unpublished data, 1994 and 1996.

BID = twice a day; BDP = Beclomethasone dipropionate; PEF = peak expiratory flow.

haler (Fig. 10) is a multidose preloaded powder system.[29] The device instructions shown in Table 3 are based on the manufacturers' instructions but incorporate our observations on their step-by-step use.

Problems with Use of Dry Powder Inhalers

The need for a high inspiratory flow to aerosolize the drug powder results in high oropharyngeal impaction. Particles depositing in the oropharynx are mostly those of the carrier, because the smaller drug particles (~2 μm in diameter) in the aerosol are less influenced by impaction than the larger carrier particles. Very few carrier particles deposit in the lower respiratory tract, and irritant effects following inhalation of the dry powder are seldom observed. Oropharyngeal side effects following inhalation of corticosteroids appear to be less common with DPIs than with pMDIs.[9,30] Clinically significant oropharyngeal *Candida* infection occurs in ~5% of patients,[31] whereas dysphonia occurs in 30–50% of patients using inhaled corticosteroids via pMDI.[32] Use of a spacer with a pMDI decreases the incidence of oropharyngeal candidiasis but not of dysphonia.[33–35] Alteration in the position of the vocal cords, and the dimensions of the glottis, when the patient inhales against the resistance of a DPI may reduce deposition of drug particles in the larynx compared to devices with lower inspiratory flow resistance (pMDIs, with or without spacers).

DPIs are breath-actuated and therefore reduce the problem of coordinating inspiration with actuation, which complicates the use of pMDIs. Table 4 lists the differences in technique for pMDIs and DPIs. Although many patients find DPIs more convenient and easier to use than pMDIs, one report suggests that up to 25% of patients may use DPIs improperly.[36] Clinicians should be well versed in the use of DPIs and prepared to provide patients repeated instruction and periodic review of technique.[37–39] The availability of a dose counter in the newer DPIs is an asset for many patients.

Comparison of Pressurized Metered Dose Inhalers and Dry Powder Inhalers

Table 5 compares the performance and convenience factors of pMDIs and DPIs. Although DPIs provide some distinct advantages over pMDIs, there could be problems with the use of DPIs in humid environments. Also, patients should not use DPIs for relief of symptoms during acute exacerbations of asthma. Bronchodilators administered via DPI can successfully treat patients with acute asthma,[40–42] but patient's using DPIs for maintenance bronchodilator therapy are advised to use a pMDI for acute symptoms, because patients with severe airway obstruction might not be able to generate the high inspiratory flow required for optimal DPI use. Further investigations are needed to establish the efficacy of DPIs in patients with acute airway obstruction.

Only a few investigations have been conducted on the effects of drugs administered via DPI versus the same drugs administered via pMDI. For maintenance therapy, long-term effects of these drugs are of greater clinical relevance than responses to administration of single doses. Table 6 summarizes the results of several studies of the long-term effects of asthma drugs administered via DPI versus via pMDI. The table indicates that the efficacy of drugs administered via DPI is similar to that observed with pMDIs, so differences in the in vitro performance of the various devices do not influence long-term responses to drug administration. However, individual responses vary, and switching a patient from a pMDI to a DPI or vice versa requires careful observation, possibly followed by dose adjustment. Very few investigations have determined equivalent doses of drugs when both the drug and the device used for inhalation therapy are changed; again, additional studies are needed.

Final Recommendations

Preferably, patients should employ only one type of aerosol-generating device for inhalation therapy. The technique of use varies among devices, and repeated instruction is highly advisable, to ensure that the patient uses the device appropriately. The use of more than one inhalation device can be confusing for patients, and may decrease their compliance with therapy. Therefore, clinicians should actively try to reduce the number of devices used by a patient. At present, DPIs can be considered as alternatives to pMDIs in patients who can generate inspiratory flow rates greater than 30–60 L/min and who are unable to use pMDIs effectively. Moreover, some patients may prefer a particular DPI. DPIs are recommended for prophylactic and maintenance therapy in patients with asthma and chronic obstructive pulmonary disease, but not for patients with acute bronchoconstriction, or children less than 6 years of age. Therefore, one drawback of DPIs is that they do not substitute for pMDIs in all clinical situations, and the appropriate dose of a given drug may be different for a pMDI and a DPI. Deciding the appropriate dose of inhaled corticosteroids can be a particularly vexing problem, since it is difficult to determine bioequivalence with corticosteroids. Further research is needed to determine equivalent dosages when both the drug and the device used are altered.

ACKNOWLEDGEMENTS

We thank Jared Young for preparing the illustrations. We also thank Glaxo Wellcome Inc, Research Triangle Park, North Carolina, for providing data on clinical studies comparing effects of drugs administered via DPIs and pMDIs.

REFERENCES

1. Ganderton D. The generation of respirable clouds from coarse powder aggregates. J Biopharm Sci 1992;3:101–105.
2. Dolovich M, Rheim R, Rashid F, Coates G, Hill M, Bowen B. Measurement of the particle size and dosing characteristics of a radiolabelled albuterol-sulphate lactose blend used in the SPIROS dry powder inhaler. In: Dalby RN, Byron P, Farr SY, editors. Respiratory drug delivery V. Buffalo Grove: Interpharm Press; 1996: 332–335.
3. Hansen OR, Pederson S. Optimal inhalation technique with terbutaline Turbuhaler. Eur Respir J 1989;2(7):637–639.
4. Engel T, Heinig JH, Madsen F, Nikander K. Peak inspiratory flow and inspiratory vital capacity of patients with asthma measured with and without a new dry powder inhaler device (Turbuhaler). Eur Respir J 1990;3(9):1037–1041.
5. Pedersen S, Hansen OR, Fuglsang G. Influence of inspiratory flow rate upon the effect of a Turbuhaler. Arch Dis Child 1990;65(3): 308–310.
6. Thorsson L, Edsbacker S, Conradson TB. Lung deposition from Turbuhaler is twice that from a pressurized metered-dose inhaler pMDI. Eur Respir J 1994;7(10):1839–1844.
7. Svartengren K, Lindestad P, Svartengren M, Philipson K, Bylin G, Camner P. Added external resistance reduces oropharyngeal deposition and increases lung deposition of aerosol particles in asthmatics. Am J Respir Crit Care Med 1995;152(1):32–37.

8. Persson G, Wiren JE. The bronchodilator response from inhaled terbutaline is influenced by the mass of small particles: a study on a dry powder inhaler (Turbuhaler). Eur Respir J 1989:2(3):253–256.
9. Hill LS, Slater AL. A comparison of the performance of two modern multidose dry powder asthma inhalers. Respir Med 1998;92(1):105–110.
10. Rajkumari NJ, Byron PR, Dalby RN. Testing of dry powder aerosol formulations in different environmental conditions. Int J Pharmac 1995;113:123–130.
11. Timsina MP, Martin GP, Van der Kolk H, Tushulzen E, Enthoben R. The effect of inhalation flow on the performance of a dry powder inhalation system. Int J Pharm 1992;81:199–203.
12. Timsina MP, Martin GP, Marriott C, Ganderton D, Yainneskis M. Drug delivery to the respiratory tract using dry powder inhalers. Int J Pharm 1994;101:1–13.
13. Hindle M, Byron PR. Dose emissions from marketed dry powder inhalers. Int J Pharmac 1995;116:169–177.
14. Kim CS, Garcia L. Delivery characteristics of albuterol powder aerosol by rotahaler. J Aerosol Med 1993;6:199–211.
15. Bisgaard H, Ifversen M, Klug B, Stamstrup K, Sumby B. Inspiratory flow rate through the Diskus/Accuhaler inhaler and Turbuhaler inhaler in children with asthma. J Aerosol Med 1995;8:100.
16. Pedersen S. Delivery systems in children. In: Barnes PJ, Grunstein MM, Leff AR, Woolcock AJ, editors. Asthma. Philadelphia: Lippincott-Raven: 1997:1915–1929.
17. Svedmyr N, Lofdahl C, Svedmyr K. The effect of powder aerosol compared to pressurised aerosol. Eur J Respir Dis 1982;63(suppl 119):81–88.
18. Fuller R. The Diskus: a new multi-dose powder device-efficacy and comparison with Turbuhaler. J Aerosol Med 1995;8(Suppl 2):S11–S17.
19. Olsson B, Asking L, Borgstrom L, Bondesson E. Effect of inlet throat on the correlation between measured fine particle dose and lung deposition. In: Dalby RN, Byron PR, Farr SY, editors. Respiratory drug delivery V. Buffalo Grove IL: Interpharm Press Inc; 1996:273–281.
20. Zainudin BM, Biddiscombe M, Tolfree SE, Short M, Spiro SG. Comparison of bronchodilator responses and deposition patterns of salbutamol inhaled from a pressurised metered dose inhaler, as a dry powder, and as a nebulised solution. Thorax 1990;45(6):469–473.
21. Borgstrom L, Newman SP. Total and regional lung deposition of terbutaline sulphate inhaled via a pressurised pMDI or via Turbuhaler. Int J Pharm 1993;97:47–53.
22. Glaxo Wellcome Inc. Diskhaler product information sheet. Glaxo Wellcome Inc, Research Triangle Park, North Carolina.
23. Tukainen H, Terho EO. Comparison of inhaled salbutamol powder and aerosol in asthmatic patients with low peak expiratory flow level. Eur J Clin Pharmacol 1985;27(6):645–647.
24. Golish J, Curtis-McCarthy P, McCarthy K, Kavuru M, Wagner W, Beck G, Eng P. Albuterol delivered by metered-dose inhaler (pMDI), pMDI with spacer, and Rotahaler device: a comparison of efficacy and safety. J Asthma 1998;35(4):373–379.
25. Borgstrom L, Derom E, Stahl E, Wahlin-Boll E, Pauwels R. The inhalation device influences lung deposition and bronchodilating effect of terbutaline. Am J Respir Crit Care Med 1996;153(5):1636–1640.
26. Newman SP, Pavia D, Clarke SW. Simple instructions for using pressurized aerosol bronchodilators. J R Soc Med 1980;73(11):776–779.
27. Lofdahl CG, Andersson L, Bondesson E, Carlsson LG, Friberg K, Hedner J, et al. Differences in bronchodilating potency of salbutamol in Turbuhaler as compared with a pressurized metered-dose inhaler formulation in patients with reversible airway obstruction. Eur Respir J 1997;10(11):2474–2478.
28. Boulet LP, Cowie R, Johnston P, Krakovsky D, Mark S. Comparison of Diskus inhaler, a new multidose powder inhaler, with Diskhaler inhaler for the delivery of salmeterol to asthmatic patients. Canadian Study Group. J Asthma 1995;32(6):429–436.
29. Pauwels RA, Hargreave FE, Camus P, Bukoski M, Stahl E. A 1-year comparison of Turbuhaler vs pressurized metered-dose inhaler in asthmatic patients. Chest 1996;110(1):53–57.
30. Selroos O, Backman R, Forsen KO, Lofroos AB, Niemisto M, Pietinalho A, et al. Local side effects during 4-year treatment with inhaled corticosteroids: a comparison between pressurized metered-dose inhalers and Turbuhaler. Allergy 1994;49(10):888–890.
31. Willey RF, Milne LJ, Crompton GK, Grant IW. Beclomethasone dipropionate aerosol and oropharyngeal candidiasis. Br J Dis Chest 1976;70(1):32–38.
32. Barnes PJ, Pedersen S. Efficacy and safety of inhaled corticosteroids in asthma. Report of a worksohp held in Eze, France, October 1992. Am Rev Respir Dis 1993;148(4 Pt 2): S1–S26.
33. Toogood JH, Baskerville J, Jennings B, Lefcoe NM, Johansson SA. Use of spacers to facilitate inhaled corticosteroid treatment of asthma. Am Rev Respir Dis 1984;129(5):723–729.
34. Williamson I, Matusiewicz S, Brown PH, Greening AP, Crompton GK. Frequency of voice problems and cough in patients using pressurized aerosol inhaled steroid preparations. Eur Respir J 1995;8(4):590–592.
35. Toogood JH, Jennings B, Baskerville J, Andersson J, Johansson SA. Dosing regimen of budesonide and occurrence of oropharyngeal complications. Eur J Respir Dis 1984;65(1):35–44.
36. Virchow JC Jr, Kroegel C, Matthys H. Antiasthma drug delivery. What is on the horizon? (review) Clin Pharmacokinet 1994;27(2):85–93.
37. Kesten S, Elias M, Cartier A, Chapman KR. Patient handling of a multidose dry powder inhalation device for albuterol. Chest 1994;105(4):1077–1081.
38. Hanania NA, Wittman R, Kesten S, Chapman KR. Medical personnel's knowledge of and ability to use inhaling devices: metered-dose inhalers, spacing chambers, and breath-actuated dry powder inhalers. Chest 1994;105(1):111–116.
39. van der Palen J, Klein JJ, Schildkamp AM. Comparison of a new multidose powder inhaler (Diskus/Accuhaler) and the Turbuhaler regarding preference and ease of use. J Asthma 1998;35(2):147–152.
40. Tonnesen F, Laursen LC, Evald T, Stahl E, Ibsen TB. Bronchodilating effect of terbutaline powder in acute severe bronchial obstruction. Chest 1994;105(3):697–700.
41. Pauwels R. Inhalation device, pulmonary deposition and clinical effects of inhaled therapy (review). J Aerosol Med 1997;10(Suppl 1):S17–S21.
42. Nana A, Youngchaiyud P, Maranetra N, Boe J, Lofdahl CG, Selroos O, Stahl E. Beta 2-agonists administered by a dry powder inhaler can be used in acute asthma. Respir Med 1998;92(2):167–172.
43. Hartley JP, Nogrady SG, Seaton A. Long-term comparison of salbutamol powder aerosol in asthmatic out-patients. Br J Dis Chest 1979;73(3):271–276.
44. Muittari A, Ahonen A. Comparison of the bronchodilator effect of inhaled salbutamol powder and pressurized salbutamol aerosol. Curr Ther Res 1979;25:804–808.
45. Bronsky E, Bucholtz GA, Busse WW, Chervinsky P, Condemi J, Ghafouri MA, et al. Comparison of inhaled albuterol powder and aerosol in asthma. J Allergy Clin Immunol 1987;79(5):741–747.
46. Lundback B, Alexander M, Day J, et al. Evaluation of fluticasone propionate (500 microg/day) administered either as dry powder via a Diskhaler inhaler or pressurized inhaler and compared with beclomethasone dipropionate (1000 microg/day) administered by pressurized inhaler. Respir Med 1993;87(8):609–620.

47. Engel T, Heinig JH, Malling HJ, Scharling B, Nikander K, Madsen F. Clinical comparison of inhaled budesonide delivered either via pressurized metered dose inhaler or Turbuhaler. Allergy 1989;44(3): 220–225.
48. Nieminen MM, Lahdensuo A. Inhalation treatment with budesonide in asthma: a comparison of Turbuhaler and metered dose inhalation with Nebuhaler. Acta Therapeutica 1995;21:179–192.
49. Togood JH, White FA, Baskerville JC, Fraher LJ, Jennings B. Comparison of the antiasthmatic, oropharyngeal, and systemic glucocorticoid effects of budesonide administered through a pressurized aerosol plus spacer or the Turbuhaler dry powder inhaler. J Allergy Clin Immunol 1997;99(2):186–193.
50. Dalby RN, Hickey AJ, Tiano SL. Medical devices for the delivery of therapeutic aerosols to the lungs. In: Hickey AJ, editor. Inhalation aerosols: physical and biological basis for therapy. Lung Biology in Health and Disease, vol 94. New York: Marcel Dekker; 1996:441–473.
51. Clark T, Rees J. Practical management of asthma. London: Martin Dunitz; 1985:134.
52. Crompton GK. Delivery systems. In: Kay AB, editor. Allergy and allergic diseases. London: Blackwell Science; 1997:1440–1450.

Bicarbonate, Oh, Bicarbonate

H Frederic Helmholz Jr MD

The source of bicarbonate in the body is the carbon dioxide resulting from metabolic energy production in the tissues. Carbon dioxide is remarkably soluble in the body. Whenever rapid acid-base exchanges take place, carbonic anhydrase is present to accelerate hydration, and the reverse. Carbonic anhydrase is present in red blood cells, gastric and intestinal mucosa, and the pancreas, for example. Equilibrium in the arterial blood of carbon dioxide partial pressure with bicarbonate ion concentration and pH is determined by the cationic concentration available to match bicarbonate and buffer protein anions. This concentration is provided by renal adjustment of ionic concentrations, and was called buffer base by Henderson[1] and by Singer and Hastings.[2] Its difference from normal was called base excess by Astrup[3] and by Anderson.[4] Figures 1, 2, and 3 show these equilibria. The sloping buffer line for arterial blood in each figure is derived by assuming that the blood proteins, though buffering fully during each circulation, are represented in arterial blood as in equilibrium with the entire body. Thus, they buffer for some 3 times the volume of the blood.

Figure 1 represents normal relationships in arterial blood. Figure 2 represents the relationship with decreased strong ion difference (acidosis). Figure 3 represents relationships with increased strong ion difference (alkalosis). Figure 4 shows the difference in pH produced by carbon dioxide uptake in the tissues, the blood providing maximum buffering (circled Point 2) and oxygen diffusion out of hemoglobin, providing still more buffering as the weaker acid, reduced hemoglobin, is formed (the Haldane effect) (circled Point 3).

The normal kidney tubules, by active transport of cations, will return "filtered bicarbonate" to the blood as carbonic anhydrase makes bicarbonate available to accompany cations. This is the process of "bicarbonate regeneration" proposed as maintaining the buffer power of the blood. In reality, the kidney is maintaining strong ion difference, and bicarbonate goes along.[6–7] The bicarbonate-carbon dioxide system provides the transport system by which acid is removed from the body. This, I suppose, is a most effective form of buffering, but it is not what chemists call the action of a "buffer."

REFERENCES

1. Henderson LJ. The Blood. New Haven CT: Yale University Press; 1928.
2. Singer RB, Hastings AB. Improved clinical method for estimation of disturbances of acid-base balance of human blood. Medicine 1948; 27:223–242.
3. Astrup P, Jorgensen K, Siggaard-Anderson O, Engel K. Acid-base metabolism: new approach. Lancet 1960;278:1035–1039.
4. Siggaard-Anderson O, Engel K, Jorgensen K, Astrup P. A micro method for determination of pH, carbon dioxide tension, base excess and standard HCO_3^- in capillary blood. Scand J Clin Lab Invest 1960;12:172–176.
5. Helmholz Jr HF. The physicochemical basis for respiratory gas exchange. In: Burton GG, Hodgkin JE, Ward JJ, eds. Respiratory care: a guide to clinical practice, 4th ed. Philadelphia: Lippincott-Raven; 1997:137–150.
6. Morfei J. Stewart's strong ion difference approach to acid-base analysis. Respir Care 1999;44(1):45–52.
7. Swenson ER. Strong ion difference approach: can a strong case be made for its use in acid-base analysis? (editorial) Respir Care 1999: 44(1)26–28.

H Frederic Helmholz Jr MD is Emeritus Staff with The Mayo Clinic, Rochester, Minnesota.

Correspondence: H Frederic Helmholz Jr MD, 600 4th Street SW, Rochester Minnesota 55902.

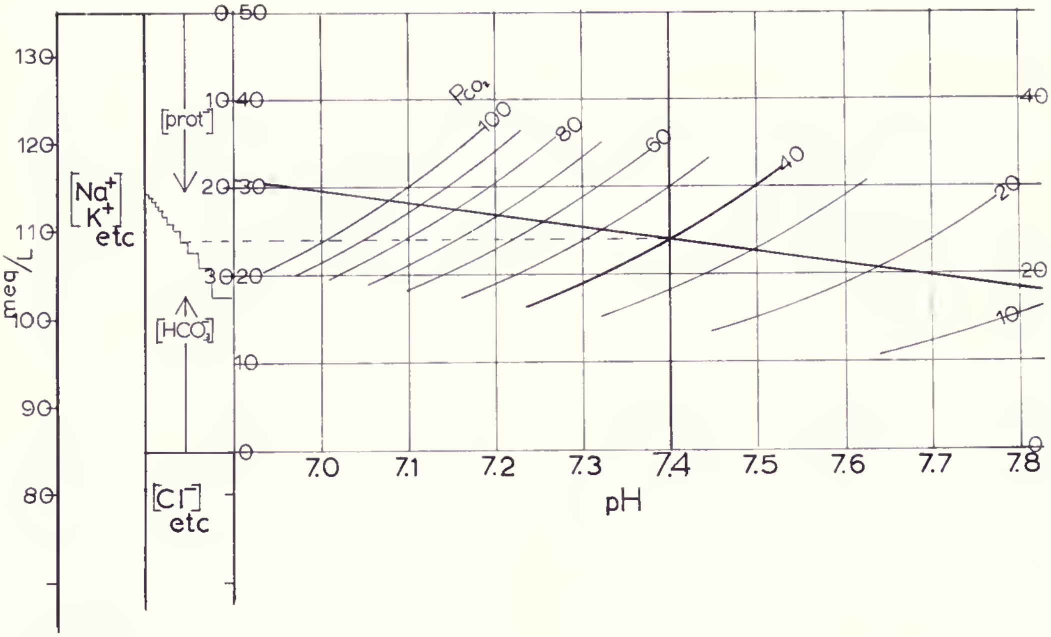


Fig. 1. Normal arterial blood. (From Reference 5, with permission.)

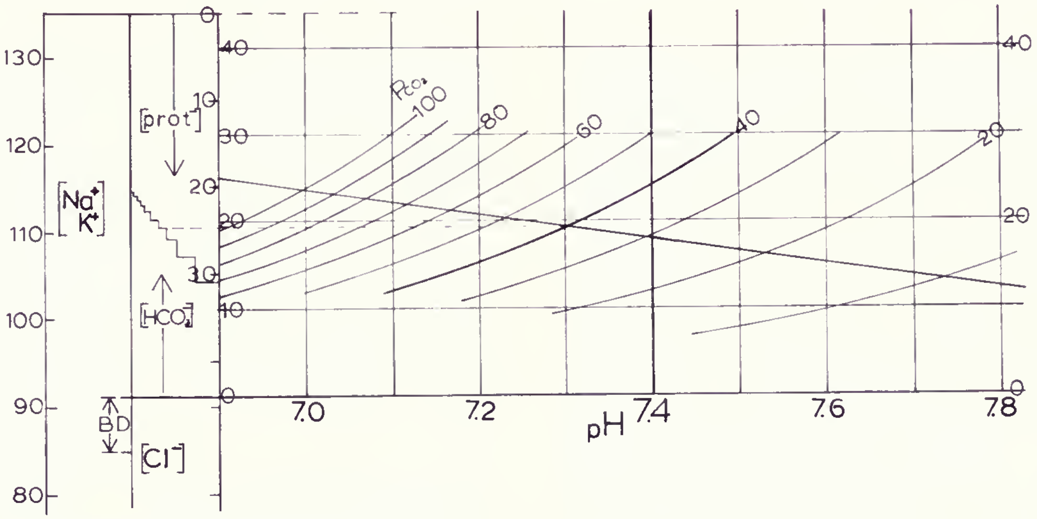


Fig. 2. Arterial blood, decreased strong ion difference (acidosis). (From Reference 5, with permission.)

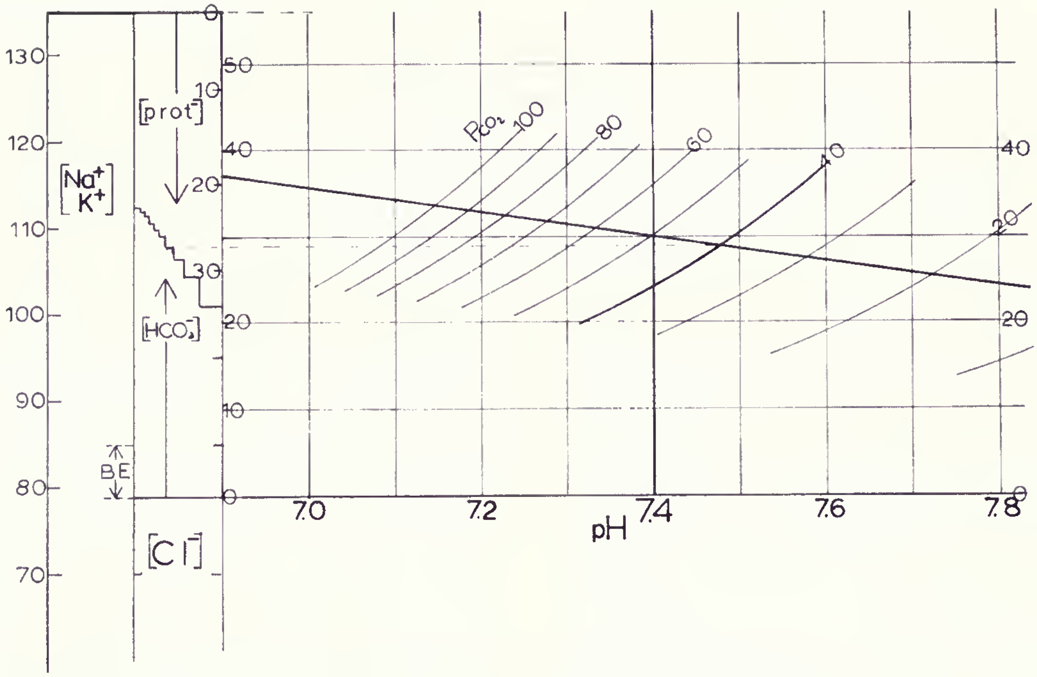


Fig. 3. Arterial blood, increased strong ion difference (alkalosis). (From Reference 5, with permission.)

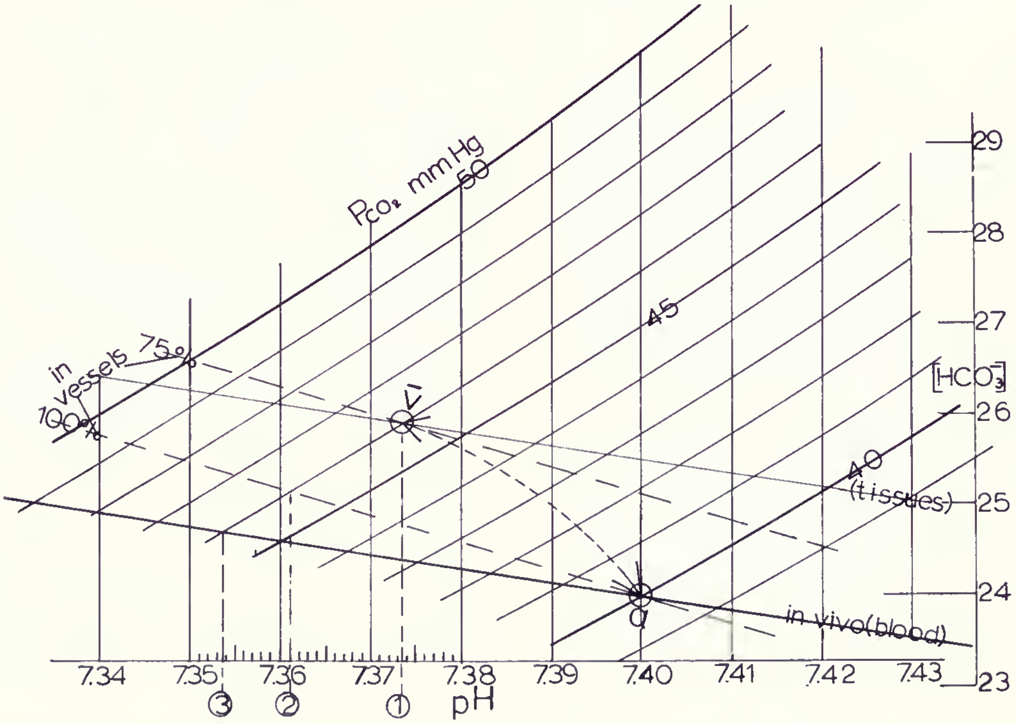


Fig. 4. Blood equilibrated with P_{CO2} of 46 mm Hg with "whole body equilibrated" blood (circled Point 3); with blood in vessels (circled Point 2); and with blood having lost oxygen (circled Point 1). (Modified from Reference 5, with permission.)

Flow-Volume Loop Analysis of Airflow Limitation: "All that Obstructs Is Not Bronchial Asthma"

Deborah A Hunton RRT RPFT and David A Kaminsky MD

Case Presentation

A 42-year-old white female was referred to the pulmonary outpatient clinic by her primary care physician for a several-week history of dyspnea and cough. She had been placed on albuterol, salmeterol, and fluticasone metered dose inhalers, but continued to have symptoms despite some improvement. She had recently been seen in the emergency department for an exacerbation of these symptoms that was unresponsive to medications at home. During that emergency room visit, her peak flow improved from 50 L/min to 130 L/min 3 hours after the initiation of nebulized albuterol and intravenous corticosteroids. By the time the patient presented to the outpatient clinic, she was finishing her third course of oral prednisone, but continued to suffer shortness of breath and paroxysms of coughing.

On presentation to the pulmonary clinic, the patient appeared comfortable, with a temperature of 37° C, blood pressure of 142/82 mm Hg, heart rate of 80 beats per minute, and respiratory rate of 16 breaths per minute. Her physical examination was notable for good air movement bilaterally, with no wheezes, crackles, or stridor. The chest x-ray report from 2 months prior was normal. Table 1 and Figure 1 show the results of routine spirometry.

Question: How would you interpret the results of the routine spirometry?

Answer: The data in Table 1 and Figure 1 show moderate airflow limitation, with significant improvement in forced expiratory volume in the first second (FEV_1) following albuterol. The shape of the pre-bronchodilator flow-volume loop suggests a component of large airway intrathoracic obstruction, but this appears to resolve following bronchodilator.

The patient was diagnosed with asthma and discharged on albuterol, salmeterol, and an increased dosage of fluticasone, and asked to return in one month. When the patient returned for that appointment, she complained of body cramps, neck tenderness, swelling in the upper sternum, difficulty "getting air in or out," and that the albuterol was providing only partial relief of her dyspnea. Vital signs were: temperature 37.4° C, blood pressure 120/64 mm Hg, heart rate 84 beats per minute, and respiratory rate 20 breaths per minute. Her physical examination revealed clear breath sounds, no wheezing, crackles, or stridor, and frequent cough. Examination of the thorax revealed an approximately 3 cm oval swelling halfway between the suprasternal notch and the larynx.

Table 2 and Figure 2A show the results of the second spirometry.

Question: How would you interpret the results of these follow-up data?

Answer: The data in Table 2 and Figure 2A show severe airflow limitation, with no significant improvement following albuterol (FEV_1 increased < 200 mL), and compared to previous testing, the FEV_1 is further reduced. The flow-volume loop now shows a persistent pattern of variable intrathoracic obstruction.

A chest x-ray (Fig. 3) revealed marked widening of the superior mediastinum, with displacement of the trachea to the right, as well as compression of the trachea indicative of a superior mediastinal mass. A chest computed tomography scan (Fig. 4) showed the mass to be 7 cm × 7 cm, that it surrounded the great vessels, and that it was significantly compressing and deviating the trachea. A fine needle aspiration of the mass revealed it to be nodular sclerosing Hodgkin's lymphoma.

Within a week of the second clinic visit, the patient's condition worsened to the point of stridor and respiratory failure. She was treated urgently with steroids and radiation therapy, and chemotherapy was started shortly thereafter. Two weeks following chemotherapy and radiation, her dyspnea and cough subsided.

Deborah A Hunton RRT RPFT is affiliated with the Department of Respiratory Therapy, Fletcher Allen Health Care, Burlington, Vermont. David A Kaminsky MD is affiliated with the Pulmonary Function Laboratory, Fletcher Allen Health Care, Burlington, Vermont, and with the Pulmonary Disease and Critical Care Medicine Unit, University of Vermont College of Medicine, Burlington, Vermont.

Correspondence: David A Kaminsky MD, Pulmonary Disease and Critical Care Medicine, Biven C-317, University of Vermont College of Medicine, Burlington Vermont 05405. E-mail: dkaminsk@zoo.uvm.edu.

Table 1. Spirometric Data before and after Administration of Albuterol during First Clinic Visit

	Pre-Albuterol (% of predicted)	Post-Albuterol (% of predicted)	% change
FVC (L)	3.89 (101)	3.89 (101)	0
FEV_1 (L)	1.80 (61)	2.20 (74)	+22
FEV_1/FVC (%)	46 (60)	57 (73)	+24

FVC = Forced vital capacity
FEV_1 = Forced expiratory volume in the first second
FEV_1/FVC = Ratio of FEV_1/FVC

Table 2. Spirometric Data before and after Administration of Albuterol during Second Clinic Visit

	Pre-Albuterol (% of predicted)	Post-Albuterol (% of predicted)	% change
FVC (L)	3.74 (97)	3.75 (98)	0
FEV_1 (L)	0.97 (33)	1.13 (38)	+16
FEV_1/FVC (%)	26 (34)	30 (39)	+15

FVC = Forced vital capacity
FEV_1 = Forced expiratory volume in the first second
FEV_1/FVC = Ratio of FEV_1/FVC

Six months after completing the chemotherapy and radiation for Hodgkin's lymphoma, the patient returned to the pulmonary clinic for routine follow-up. She was off all inhalers and had no respiratory symptoms. Physical examination was unremarkable. Spirometry revealed not only that the forced vital capacity (FVC) and FEV_1 had nearly normalized, but also that the variable intrathoracic obstruction had completely resolved (Fig. 2B).

Discussion

This case clearly illustrates the importance of the shape of the flow-volume loop. All too often, pulmonary function labs report numerous indices derived from simple spirometry, but fail to show the flow-volume loop itself. The shape of the loop may give valuable information about the site and extent of airflow limitation.[1-4] Airflow limitation can occur on inspiration or expiration, and can be variable or fixed throughout the respiratory cycle.[1] Misinterpreting the flow-volume loop as normal when in fact the loop suggests upper airway obstruction is among the more frequent errors made by general internists in spirometry interpretation.[5]

The inspiratory loop can be a sensitive indicator of extrathoracic abnormalities that limit inspiratory flow, such as vocal cord dysfunction.[6] While the inspiratory loop is important, the more common finding of expiratory loop abnormalities due to intrathoracic airflow limitation must not be assumed to be due to asthma or chronic obstructive pulmonary disease alone. Indeed, close inspection of the initial flow-volume loop (Fig. 1A), reveals an initial sharp peak flow, followed by an abrupt plateau of flow, then a gradual decline in flow as the lung approaches residual

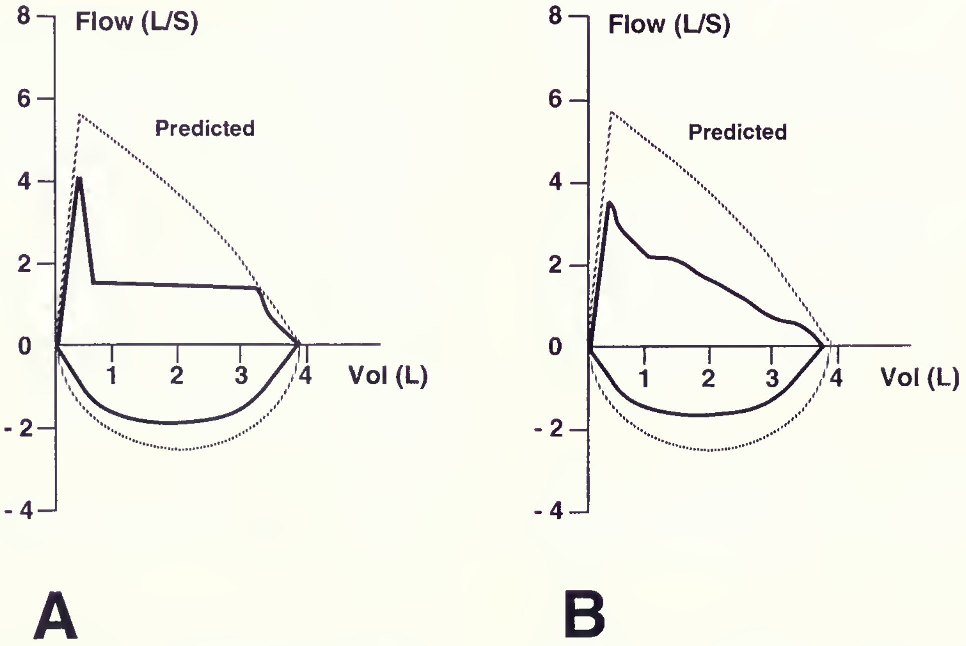


Fig. 1. Flow-volume loops (solid line) before (A) and after (B) the administration of the bronchodilator albuterol, during the first clinic visit. The dashed line indicates the predicted flow-volume relationship.

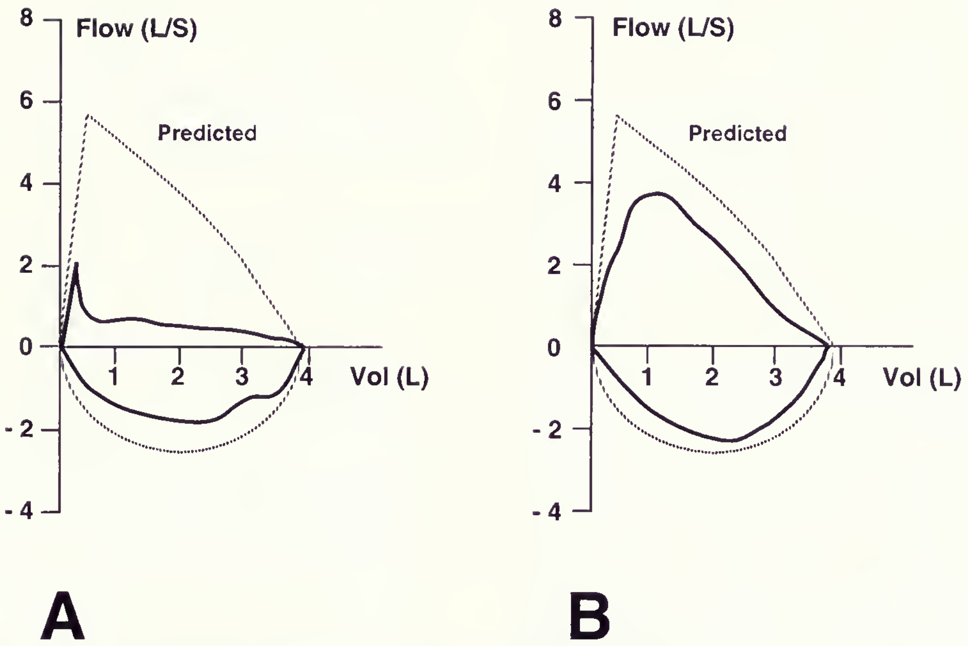


Fig. 2. Flow-volume loop (solid line) during the second clinic visit (A), and six months following completion of radiation and chemotherapy for Hodgkin's lymphoma (B). The dashed line indicates the predicted flow-volume relationship.

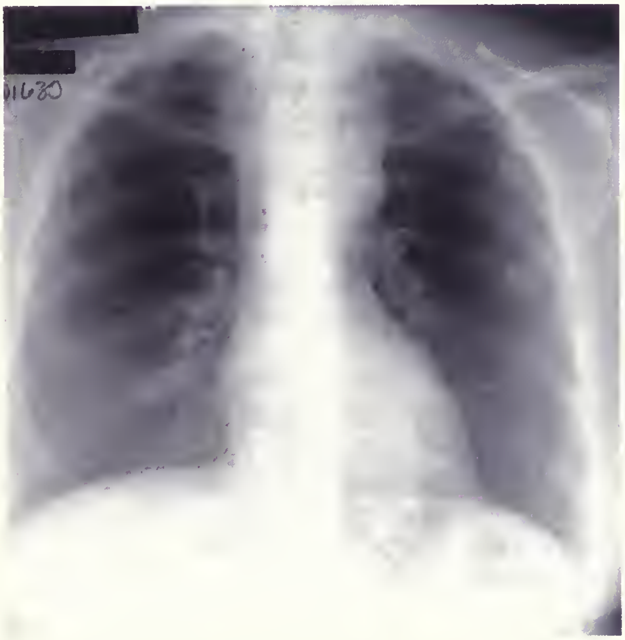


Fig. 3. Posterior-anterior chest radiograph showing widening of the mediastinum, associated with tracheal narrowing and shift to the right.

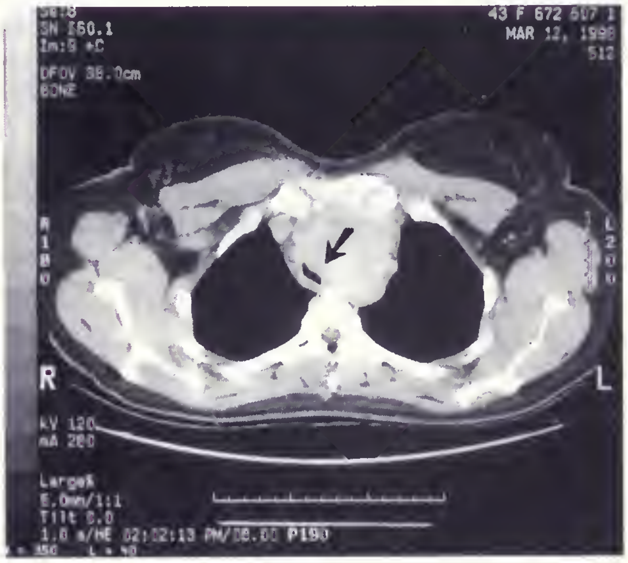


Fig. 4. Computed tomography image of the chest at the level of the mediastinum. The figure depicts a cross-sectional image of the thorax as the patient is lying on her back with her feet toward the reader. Similar to a plain chest radiograph, the lungs appear dark, surrounded by the soft tissues and bones (in white). The arrow is on the tumor mass in the mediastinum, and points toward the trachea, which is highly compressed and deviated to the right.

volume. This is not the usual morphology of the upward concavity of expiratory flow seen in typical asthma or chronic obstructive pulmonary disease.[7,8] The abrupt plateau suggests the possibility of another physical cause of intrathoracic airflow obstruction. At high lung volumes, the obstruction would be partially relieved by the dilation of the involved airway, allowing a peak flow to occur. But as the lung emptied, the obstruction would abruptly limit flow to a fixed level, until the lung emptied further and flow became limited by dynamic airway narrowing.[9] This latter phenomenon is seen as the gradual decline in flow at the terminal portion of the expiratory loop, abutting the predicted maximal expiratory flow envelope.

In this case, the patient had some reversal of airflow limitation with bronchodilator, which improved mid-expiratory flows to such an extent as to almost completely eliminate the plateau in the pre-bronchodilator studies. This reversal is certainly consistent with asthma, and is what led the clinicians involved in the case to accept asthma as the sole initial diagnosis. In addition, the post-bronchodilator loop had assumed a configuration somewhat similar to that described by Tien et al[10] as a normal variant in young, healthy individuals. This configuration is characterized by a "bump" in the mid-portion of the expiratory limb that is concave to the volume axis. The current case, however, clearly shows diminished flow rates for any given lung volume, and therefore cannot be considered a variation of normal.

The ultimate diagnosis in this case was only determined after the patient returned with worsening symptoms, identification of unambiguous physical findings, and the persistence of the severe plateau in flow following bronchodilator (Fig. 2A). The near-complete normalization of the flow-volume loop following treatment (Fig. 2B) confirmed that the predominant initial abnormality was airflow obstruction caused by the intrathoracic lymphoma. However, given the clinical history of airflow improvement following bronchodilator treatment during the initial visit, asthma was probably also present.

This case illustrates the importance of closely inspecting the flow-volume loop, the shape of which is determined not only by the degree of airflow limitation, but also by the anatomic nature and location of obstructing processes. Although such information may be subtle, it must not be overlooked if the pulmonary function technologist and physician are to realize the full diagnostic potential of the flow-volume loop.

REFERENCES

1. Miller RD, Hyatt RE. Obstructing lesions of the larynx and trachea: clinical and physiologic characteristics. Mayo Clinic Proc 1969;44(3): 145–161.
2. Miller RD, Hyatt RE. Evaluation of obstructing lesions of the trachea and larynx by flow-volume loops. Am Rev Respir Dis 1973;108(3): 475–481.
3. Kryger M, Bode F, Antic R, Anthonisen N. Diagnosis of obstruction of the upper and central airways. Am J Med 1976;61(1):85–93.
4. Guntupalli KK Bandi V, Sirgi C, Pope C, Rios A, Eschenbacher W. Usefulness of flow volume loops in emergency center and ICU settings. Chest 1997;111(2):481–488.
5. Hnatiuk O, Moores L, Loughney T, Torrington K. Evaluation of internists' spirometric interpretations. J Gen Intern Med 1996;11(4): 204–208.
6. Newman KB Mason UG 3d, Schmaling KB. Clinical features of vocal cord dysfunction. Am J Respir Crit Care Med 1995;152(4 Pt 1):1382–1386.
7. Kapp MC, Schachter EN, Beck GJ, Maunder LR, Witek TJ Jr. The shape of the maximum expiratory flow volume curve. Chest 1988; 94(4):799–806.
8. Mead J. Analysis of the configuration of maximum expiratory flow-volume curves. J Appl Physiol 1978;44(2):156–165.
9. Mead J, Turner JM, Macklem PT, Little JB. Significance of the relationship between lung recoil and maximum expiratory flow. J Appl Physiol 1967;22(1):95–108.
10. Tien YK, Elliott EA, Mead J. Variability of the configuration of maximum expiratory flow-volume curves. J Appl Physiol 1979;46(3): 565–570.

An Obese Patient Referred for Preoperative Pulmonary Clearance

Salim Kathawalla MD and Muzaffar Ahmad MD

Case Summary

A 53-year-old moderately obese (body mass index = 39 kg/m^2) male was referred for pulmonary assessment before undergoing a left shoulder implant for painful osteoarthritis. He gave a history of hypertension, cigarette smoking for 90 pack-years, and symptoms of exertional dyspnea and mild hypersomnolence. Physical examination and chest radiography were normal. Table 1 shows spirometry results, and Figure 1 shows the flow-volume loop. The patient underwent a diagnostic test before he was cleared for surgery.

1. What abnormality does the flow-volume loop show?
2. What is the differential diagnosis for the abnormalities in the flow-volume loop?
3. What diagnostic test was performed?

Discussion

The flow-volume loop reveals 2 abnormal features: (1) The presence of flow oscillations (the "saw-tooth sign") on the inspiratory and expiratory loops, and (2) the ratio of mid-expiratory flow (FEF$_{50\%}$) to mid-inspiratory flow (FIF$_{50\%}$) is greater than 1.

The saw-tooth sign was first described by Sanders et al in obstructive sleep apnea (OSA).[2] It consists of reproducible oscillations occurring at constant interval on the forced expiratory and/or inspiratory part of the flow-volume loop. This pattern is caused by fluttering tissue in the upper airways of patients with OSA, which was confirmed in this patient by direct visualization using a fiberoptic bronchoscope. Though initially described in patients with OSA, saw-tooth sign can also occur in normal individuals, patients with extrathoracic airway obstruction or excess airway secretions, burn victims at risk for airway obstruction, and in patients with Parkinson's disease.[3–6]

In asymptomatic patients, the sensitivity of this sign for OSA is approximately 14–33% and specificity is 67–95%[7,8] In symptomatic snorers, the sensitivity of the sign increases to 72%. The sensitivity is increased if the flow-volume loop is performed in the supine position.[9] Sleep apnea patients with the saw-tooth sign have a higher apnea index and lower nocturnal minimum oxygen saturation than those without the saw-tooth sign. In a review of 2,800 flow-volume loops performed in a pulmonary function laboratory, the saw-tooth sign was identified in 40 subjects (1.43%). Of the 40 subjects with the saw-tooth sign, 23% had OSA, and an additional 40% had either a structural disorder of the upper airway or neurologic disease involving the upper airway muscles.[10]

Table 1. Spirometry Results

Test	Predicted	Measured	% of predicted
FVC (L)	4.90	4.17	85
FEV$_1$(L)	3.89	3.39	87
FEV$_1$/FVC	0.79	0.81	102
PEF (L/s)		7.47	
FEF$_{50\%}$		5.32	
FEF$_{25-75\%}$ (L/s)	3.75	3.70	99
ETT		7.72	
PIF (L/s)		4.24	
FIF$_{50\%}$ (L/s)		4.22	

Predicted = Mean predicted values as per Crapo et al[1]
FVC = Forced vital capacity
FEV$_1$ = Forced expiratory volume in the first second
FEV$_1$/FVC = Ratio of FEV$_1$ to FVC
PEF = Peak expiratory flow (in liters/second)
FEF$_{50\%}$ = Forced expiratory flow at 50% of the FVC
FEF$_{25-75\%}$ = Forced expiratory flow between 25% and 75% of the FVC
ETT = Exercise tolerance test
PIF = Peak inspiratory flow (in liters/second)
FIF$_{50\%}$ = Forced inspiratory flow at 50% of the FVC.

Salim Kathawalla MD was affiliated with the Department of Pulmonary and Critical Care Medicine, The Cleveland Clinic Foundation, Cleveland, Ohio at the time of this study, and is now affiliated with the Department of Pulmonary, Critical Care and Sleep Medicine, Park Nicollet Clinic, Minneapolis, Minnesota. Muzaffar Ahmad MD is affiliated with the Department of Pulmonary and Critical Care Medicine, The Cleveland Clinic Foundation, Cleveland, Ohio.

Correspondence: Salim Kathawalla MD, Department of Pulmonary, Critical Care, and Sleep Medicine, Park Nicollet Clinic, 3800 Park Nicolet Blvd, Minneapolis, MN 55416.

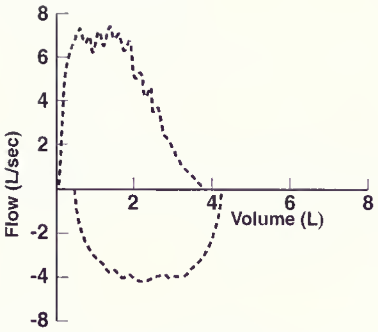


Fig. 1. Flow-volume loop on the patient.

A $\text{FEF}_{50\%}$ / $\text{FIF}_{50\%}$ ratio greater than 1 is a nonspecific sign seen in snorers and patients with upper airway abnormalities such as extrathoracic airway obstruction and sleep apnea. The ratio has a sensitivity and specificity less than the saw-tooth sign in predicting OSA.

Based on the presence of hypersomnolence, hypertension (which can be caused by OSA), and snoring, and on the appearance of the flow-volume loop, OSA was strongly suspected in this patient, and a polysomnogram confirmed severe OSA. The patient was treated with nasal continuous positive airway pressure and cleared for surgery.

Though the saw-tooth sign has limited value as a screening test for OSA, its presence on the flow-volume loop in symptomatic patients is useful in suggesting OSA.

REFERENCES

1. Crapo RO, Morris AH, Gardner RM. Reference spirometric values using techniques and equipment that meet ATS recommendations. Am Rev Respir Dis 1981;123(6):659–664.
2. Sanders MH, Martin RJ, Pennock BE, Rogers RM. The detection of sleep apnea in the awake patient. The 'saw-tooth' sign. JAMA 1981; 245(23):2414–2418.
3. Nagai A, Matsumiya H, Hayashi M, Kanemura T, Yasui S, Konno K. Lesions of the arytenoid region in a patient with exertional dyspnoea. Eur Respir J 1993;6(7):1065–1066.
4. Jubran A, Tobin MJ. Use of flow-volume curves in detecting secretions in ventilator-dependent patients. Am Respir Crit Care Med 1994;150(3):766–769.
5. Schiffman PL. A "saw-tooth" pattern in Parkinson's disease. Chest 1985;87(1):124–126.
6. Haponik EF, Munster AM, Wise RA, Smith PL, Meyers DA, Britt EJ, Bleecker ER. Upper airway function in burn patients. Correlation of flow-volume curves and nasopharyngoscopy. Am Rev Respir Dis 1984;129(2):251–257.
7. Rauscher H, Popp W, Zwick H. Flow-volume curves in obstructive sleep apnea and snoring. Lung 1990;168(4):209–214.
8. Katz I, Zamel N, Slutsky AS, Rebuck AS, Hoffstein V. An evaluation of flow-volume curves as a screening test for obstructive sleep apnea. Chest 1990;98(2):337–340.
9. Shore ET, Millman RP. Abnormalities in the flow-volume loop in obstructive sleep apnea sitting and supine. Thorax 1984;39(10):775–779.
10. Vincken W, Cosio MG. Flow oscillations on the flow-volume loop: a nonspecific indicator of upper airway dysfunction. Bull Eur Physiopathol Respir 1985;21(6):559–567.

A 38-Year-Old Man with Progressive Dyspnea and a Clear Chest Roentgenogram

Robert J Schilz DO PhD

Case Summary

A 38-year-old man presents with a one-year history of progressive dyspnea, initially only with running, but recently with climbing one flight of stairs. Medical history was remarkable only for non-A, non-B, non-C hepatitis without chronic liver disease, and remote intravenous drug use. The patient is a nonsmoker. His sister had similar complaints and ultimately underwent lung transplantation. Physical examination revealed no adventitious lung sounds. Chest roentgenogram showed clear lung fields with prominent hila, and a suggestion of peripheral pruning of the blood vessels. A perfusion scan of the lung showed no defects. Arterial blood measurements on room air were: pH 7.47; arterial carbon dioxide tension (P_{aCO_2}) 28 mm Hg; arterial oxygen tension (P_{aO_2}) 52 mm Hg; bicarbonate 21 mEq/L; O_2 saturation 84%. Table 1 shows the results of pulmonary function testing.

1. What is your diagnosis?

Discussion

The marked decrease in diffusing capacity (DL_{CO}) with preserved flow rates and lung volumes suggests the possibility of pulmonary vascular disease. Marked hypoxemia on room air with polycythemia is also noted. General pathophysiologic processes that can result in a decreased DL_{CO} include:

1. Decreased oxygen carrying capacity, especially anemia.
2. Ventilation-perfusion mismatch.
3. Surgical loss of lung parenchyma and its accompanying capillary bed.
4. Destruction of normal alveolar parenchyma.
5. Compromise or obliteration of the pulmonary vascular bed.

Closer examination of the data in this case also reveals polycythemia, which would tend to increase rather than decrease DL_{CO}. Neither anemia nor surgical loss of lung parenchyma would be expected to produce significant hypoxemia if the underlying or remaining lung were free of disease. In this case, there were no historical or physical examination findings suggesting parenchymal lung disease. The chest x-ray showed no significant parenchymal abnormality, which would be expected on a good quality x-ray given this degree of diffusion impairment. The radiographic evidence of enlarged hilar vessels with distal pruning suggest primary pulmonary hypertension (PPH).

In the absence of parenchymal changes, pulmonary vascular disease is a likely explanation for the diffusion abnormalities. Several pathologies can reduce the surface area of the vascular bed (and DL_{CO}), including vasculitis, thromboembolism, cardiac valvular disease, chronic pressure of volume overload, or hypertension, suggested by the plain radiograph.[2]

In this case, ventilation-perfusion scanning failed to yield an intermediate or high probability scan, which would be expected in the syndrome of chronic thromboembolic pulmonary hypertension.[3] There was no evidence of a systemic inflammatory condition or vasculitis. Intravenous drug use may involve injection of talc, which could lead to an obliterative process.[4] Human immunodeficiency virus infection[5] or hepatitis with portal hypertension[6] acquired from sharing contaminated syringes can lead to syndromes indistinguishable from PPH. In this patient, the absence of liver disease or HIV infection and the presence of a familial syndrome suggests PPH. Subsequent echocardiography indicated elevated right ventricular pressures in the absence of intracardiac shunt or valvular abnormality—findings confirmed by cardiac catheterization. The diagnosis was PPH.

PPH is a rare disorder. The incidence of PPH in the general population is approximately 1–2 per million. Familial PPH represents approximately 3–5% of the total cases of PPH. A putative genetic locus for this disorder has been mapped to chromosome 2.[7] Many patients with PPH will have a reduction in DL_{CO}. For example, the French PPH registry reported a mean DL_{CO} of 68 ± 24% predicted.[8]

Robert J Schilz DO PhD is affiliated with the Department of Pulmonary and Critical Care Medicine, The Cleveland Clinic Foundation, Cleveland, Ohio.

Correspondence: Robert J Schilz DO PhD, Department of Pulmonary and Critical Care Medicine, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland OH 44106.

Table 1. Spirometry Results

Test	Predicted	LLN	Measured	% Predicted
FVC (L)	5.41	4.29	5.73	106
FEV_1 (L)	4.34	3.49	3.92	90
FEV_1/FVC	0.80	0.72	0.68	85
TLC (L)	7.35	5.74	7.42	101
DL_{CO} (mL/mm Hg/s)	27.0	22.0	6.84	25
DL/VA	5.21	4.09	0.93	18
Hemoglobin			18.2	

Predicted = Mean predicted values as per Crapo et al[1]
LLN = Lower limit of normal, the value as the lower 5th percentile of the normal range
FVC = Forced vital capacity
FEV_1 = Forced expiratory volume in 1 second
FEV_1/FVC = Ratio of FEV_1 of FVC
TLC = Total lung capacity by helium dilution (liters)
DLCO = Diffusing capacity for carbon monoxide (mL/mm Hg/second)
DL/VA = Ratio of diffusing capacity to alveolar volume.

A decreased diffusing capacity is correlated with a worse prognosis in PPH.[9] However, patients with significant pulmonary hypertension can have a measured DL_{CO} in the normal range,[10] so the absence of significant DL_{CO} abnormalities does not exclude the PPH diagnosis. Treatment for PPH includes: anticoagulation, vasodilator therapy, and a consideration of lung transplantation. The DL_{CO} may not change with medical management of PPH.[11]

This patient was treated with oxygen, Coumadin, and intravenous prostacyclin therapy.

REFERENCES

1. Crapo RO, Morris AH, Gardner RM. Reference spirometric values using techniques and equipment that meet ATS recommendations. Am Rev Respir Dis 1981;123(6):659–664.
2. Crapo RO, Forster RE 2d. Carbon monoxide diffusing capacity. Clin Chest Med 1989;10(2):187–198.
3. Worsley DF, Palevsky HI, Alavi A. Ventilation-perfusion lung scanning in the evaluation of pulmonary hypertension. J Nucl Med 1994; 35(5):793–796.
4. Farber HW, Falls R, Glauser FL. Transient pulmonary hypertension from the intravenous injection of crushed, suspended pentazocine tablets. Chest 1981;80(2):178–182.
5. Mette SA, Palevsky HI, Pietra GG, Williams TM, Bruder E, Prestipino AJ, et al. Primary pulmonary hypertension in association with human immunodeficiency virus infection: a possible viral etiology for some forms of hypertensive pulmonary arteriopathy. Am Rev Respir Dis 1992;145(5):1196–1200.
6. Bellocq A, Milleron B. [Pulmonary hypertension: a rare complication of hepatic cirrhosis.] Rev Pneumol Clin 1993;49(4):194–196.
7. Morse JH, Jones AC, Barst RJ, Hodge SE, Wilhelmsen KC, Nygaard TG. Mapping of familial primary pulmonary hypertension locus (PPH1) to chromosome 2q31–q32. Circulation 1997;95(12):2603–2606.
8. Brenot F, Hervé P, Rain B, Simonneau G. [Primary pulmonary artery hypertension.] Rev Prat 1991;41(17):1560–1567.
9. D'Alonzo GE, Barst RJ, Ayres SM, Bergofsky EH, Brundage BH, Detre KM, et al. Survival in patients with primary pulmonary hypertension: results from a national prospective registry. Ann Intern Med 1991;115(5):343–349.
10. Dantzker DR. Primary pulmonary hypertension: the American experience. Chest 1994;105(2 Suppl):26S–28S.
11. Thurm CA, Wigley FM, Dole WP, Wise RA. Failure of vasodilator therapy to alter pulmonary diffusing capacity in systemic sclerosis. Am J Med 1991;90(5):547–552.

Letters

Letters addressing topics of current interest or material in Respiratory Care will be considered for publication. The Editors may accept or decline a letter or edit without changing the author's views. The content of letters as published may simply reflect the author's opinion or interpretation of information—not standard practice or the Journal's recommendation. Authors of criticized material will have the opportunity to reply in print. No anonymous letters can be published. Type letter double-spaced, mark it "For Publication," and mail it to Respiratory Care, 600 Ninth Avenue, Suite 702, Seattle WA 98104. Letters may also be submitted electronically at www.rcjournal.com/online_resources.

Role of Bicarbonate Ion Concentration in Acid-Base Balance

I have had the privilege of hearing Joe Morfei give verbal presentations on several occasions. His skill in synthesizing formidably complex subjects is enviable, and this skill is evident in his review that recently appeared in this Journal.[1] Joe succeeded in making an intrinsically difficult topic, Stewart's "Strong Ion Difference" concept, clear and coherent. I take issue with one of the underlying assumptions of the analysis, but my quarrel is with Stewart, and not with Morfei.

Stewart's thesis is crucially linked to the claim that acid-base balance is related to 3—and only 3—independent variables: (1) total weak acid concentration; (2) carbon dioxide tension; and (3) the so-called "strong ion difference," defined as the net electrostatic charge of the strong ions. In this schema, bicarbonate ion concentration is relegated to the status of a dependent variable. The rationale that Stewart employs to explain the dependent role of bicarbonate relates to his (entirely correct) observation that the ultimate origin of every bicarbonate ion in solution is the antecedent dissociation of carbonic acid. Indeed, if the prevailing concentration of bicarbonate were rigidly linked to the dissociation reaction of carbonic acid, this logic would be irrefutable. But the instantaneous concentration of bicarbonate at any given time is only remotely related to the source of formation of bicarbonate, but intimately linked to the kinetic adjustment and regulation of those innumerable ions long after their formation.

Unfortunately, Stewart's analysis overlooks a tremendously important source of perturbation to the body's buffering system, which flies in the face of any attempt to consider the abundance of bicarbonate to be slavishly dependent on the carbonic acid dissociation reaction. The source of that perturbation is the kidney's role as it relates to the reabsorption of bicarbonate at the distal renal tubule. Indeed, if renal reabsorption were absent, the prevailing bicarbonate ion concentration in the blood and body tissues would necessarily be immensely lower than that which prevails in health. In fact, the desperately low levels of bicarbonate that would be imposed upon the human organism in such a circumstance would not be consistent with survival, unless the body's equilibrium system were broadened to include categories of anionic/cationic species that are not associated with carbon-based life forms as we know them. In the absence of the renal regulatory machinery, the ratio of bicarbonate ion concentration to hydrogen ion concentration would be approximately 1 to 1, whereas, in actuality, that ratio (in homeostasis) is roughly 600,000 to 1. Stated another way, the effectiveness of the kidney in upregulating bicarbonate ion concentration renders the instantaneous concentration of that chemical species a full-fledged independent variable. After all, we certainly wouldn't consider iatrogenic changes in bicarbonate ion (such as occur with the infusion of sodium bicarbonate) to represent a change in a dependent variable, because this action constitutes an obvious alteration in the endogenous status of the equilibrium system from outside the body. Similarly, the continuous renal adjustment of bicarbonate ion concentration that occurs in homeostatic and nonhomeostatic conditions is fully analogous to the iatrogenic modification just invoked. Although the adjustment we're describing occurs inside the body, its effects are imposed on the equilibrium system from outside of that system.

When the irrelevant assumption of the 'dependent' nature of bicarbonate that Stewart's theory necessitates is laid aside, the Strong Ion Difference approach seems to conceal more than it reveals. This is not to say that a thoughtful consideration of the Strong Ion Difference concept is an intellectual waste of time. On the contrary, Morfei's cogent and thought-provoking review of this intriguing thesis serves to focus our attention on the operation of a wondrously and consummately effective physiologic system, the sodium bicarbonate/carbonic acid buffer system. Surely, any mental exercise that prompts us to ponder the interactions of a system to which we literally owe our very lives must be regarded as time well spent.

Bob Demers RRT
Demers Consulting Services
Carmel, California

REFERENCE

1. Morfei J. Stewart's strong ion difference approach to acid-base analysis. Respir Care 1999;44(1):45–52.

The author responds:

I would agree with Mr Demers that the human organism is a marvelously integrated physiochemical system, but must disagree with his conclusion that the renal regulatory mechanisms qualify bicarbonate as an independent variable thus refuting Stewart's Strong Ion Difference approach to acid base balance.

A function of the kidney is H^+ excretion with the concomitant active transport of Na^+ from the glomerular filtrate into the tubular cells, where it is reabsorbed back into plasma. Chloride ion passively follows sodium in order to maintain electroneutrality. This mechanism accounts for about 80% of reabsorbed sodium. The remaining 20% of reabsorbed sodium, also exchanged for H^+ across the renal cell membrane, is passively accompanied by bicarbonate ion. It is interesting to note that this bicarbonate, which has been filtered in the glomerulus as $NaHCO_3$, is not reabsorbed as a bicarbonate ion per se, but goes through the "dehydration reaction" ($HCO_3^- + H^+ \rightarrow H_2CO_3 \rightarrow CO_2 + H_2O$), combining with a secreted H^+ and ultimately ending up as a carbon dioxide and a water molecule. CO_2 moves into renal cells in response to a pressure gradient, where it goes back through the hydration reaction in the presence of carbonic anhydrase and reforms a "new" bicarbonate ion, which then electrically balances a Na^+. *The rate of absorption of bicarbonate is directly proportional to the* P_{CO_2}.[1] To paraphrase Mr Demers, bicarbonate *is* slavishly dependent on the carbonic acid dissociation reaction. The kidney also helps regulate the K^+ concentration by secretion in somewhat inverse proportion to the rate of H^+ secretion. Through the regulation of sodium, chloride, and potassium, the kidney is able to adjust the strong ion difference.

Mr Demers does raise a good question as to why the concentration of bicarbonate ion is approximately 600,000 times greater than the hydrogen ion concentration. One would think that if bicarbonate were linked to carbon dioxide via the hydrolysis reaction, then the hydrogen ion concentration should be at least as great as that of the bicarbonate, since it is produced 1 for 1 as a result of the dissociation of carbonic acid. However, at homeostasis, the cells of the human organism function best in a chemically alkaline environment, at a pH of 7.40. This, of course, means that there needs to be more base than acid in order to maintain this pH. At pH of 7.4 the bicarbonate ion concentration is normally 24 mM/L, the concentration of hydrogen ion is 39 nmol/L, and the ratio of base to acid is about 600,000 to 1. So what happens to all of the excess acid that is produced in our body each day? About 70 mEq/d are eliminated via the kidney, another 13,000 mEq/d are excreted through the lungs, and the body has several other buffer systems, including hemoglobin, plasma proteins, and phosphate, which are all capable of buffering out large quantities of H^+. This is a rather simplistic explanation of a complex physiochemical system, but I believe it answers the excellent questions Mr Demers raised about Stewart's approach to acid-base balance.

In conclusion, it should be clear that bicarbonate ion is not an independent variable, and is dependent on the concentration of dissolved carbon dioxide. Furthermore, I believe that Stewart's Strong Ion Difference approach to acid base balance requires more clinically intensive review than it has thus far received.

Joe Morfei MS RRT
Department of Cardiorespiratory Sciences
State University of New York
Health Science Center
Syracuse, New York

REFERENCE

1. Davenport HW. The ABC of acid-base chemistry, 6th ed. Chicago: The University of Chicago Press;1974:89.

The Official Journal of the American Assocation for Respiratory Care
RESPIRATORY CARE
online
www.rcjournal.com • www.rcjournal.com • www.rcjournal.com

Listing and Reviews of Books and Other Media. Note to publishers: Send review copies of books, films, tapes, and software to Respiratory Care, 600 Ninth Avenue, Suite 702, Seattle WA 98104.

Books, Films, Tapes, & Software

Understanding Acid-Base, Benjamin Abelow MD. Soft-cover, illustrated, 333 pages plus appendix. Baltimore: Williams & Wilkins; 1998. $24.95.

This book brings together the chemical, physiological, and pathological aspects of acid-base balance. The table of contents and index make all aspects available, and self-assessment quizzes are provided, so the book is useful to respiratory therapists, medical students, residents, and physicians.

The subjects treated are pertinent and, in most cases, complete; however, the author presents the concept that bicarbonate is the body's primary buffer, and devotes only 4 pages to the nonbicarbonate buffers—the proteins. This is a major weakness, since it is the proteins—plasma proteins and hemoglobin—that buffer the major acid produced by the body, carbon dioxide (CO_2). With the help of carbonic anhydrase in the red blood cells, carbonic acid is produced and buffered by the proteins. The proteins respond to pH shift by associating and freeing cations to match the bicarbonate, and provide for efficient CO_2 transport. The reverse reaction takes place in the lungs, in response to the lower partial pressure of carbon dioxide there.

Less easy to understand is that the so-called bicarbonate regeneration is really reabsorption of sodium with bicarbonate made available by the carbonic anhydrase in renal tubules. The author seems to occasionally forget the law of electro-neutrality, and thus does not bring out the effect of strong ion difference on bicarbonate concentration at any given tension of carbon dioxide. (Strong ions are those the concentration of which is not affected by changes in pH in the physiologic range.) It is this difference in concentration that represents the cationic concentration available to match protein anions and bicarbonate, and that Henderson and others called the buffer base and, in more recent literature, base excess.[1] The addition of a strong acid reduces the strong ion difference, while the addition of the salt of a weak acid such as sodium bicarbonate increases the strong ion difference. This, as Stewart points out, is fundamental to acid-base physiology. It is the kidney that adjusts difference of strong ions by selectively excreting into or reabsorbing from the glomerular filtrate.

The clinical chapters (3, 4, and 5) provide valuable discussions of the pathophysiology of and therapies for various acid-base abnormalities. The appendix is also very useful—pertinent references are discussed, including reports by Stewart, who first emphasized the concept of strong ion difference to explain buffer base and the base excess.

The book is a paperback of medium format, and is easy to use. The subjects are easy to find, the illustrations are clear, and I found no typographical errors. The author shines through as a dedicated teacher. I applaud his effort, even though I disagree with some of his interpretations.

H Frederic Helmholz Jr MD
Staff Emeritus
The Mayo Clinic
Rochester, Minnesota

REFERENCE

1. Helmholz Jr HF. The Physicochemical basis for respiratory gas exchange. In: Burton GG, Hodgkin JE, Ward JJ, editors. Respiratory care: a guide to clinical practice, 4th ed. Philadelphia: Lippincott-Raven; 1997:137–150.

Manual of Pulmonary Function Testing, 7th ed, Greg L Ruppel Med RRT RPFT. Soft-cover, illustrated. 364 pages. St Louis: Mosby-Year Book; 1998. $36.00.

This book truly has two personalities. On the one hand, it is a treasure trove of information regarding pulmonary function testing (PFT) and all its aspects. Indeed, this would be the "Bible" to study prior to taking the CPFT or RPFT exam; it alone would probably suffice as preparation for either exam. Each chapter begins with a set of objectives that clearly establish what will be covered and what the reader should be able to do upon completion of that chapter. Each chapter contains highlighted areas on interpretive strategies and criteria for acceptability for each test or procedure discussed; there are also at least one illustrative case study and self-study questions. Obviously much thought went into creating this seventh edition. On the other hand, there are glaring errors and some editorial irritations such as numerous disagreements between subjects and verbs.

In Chapter 1, "Indications for Pulmonary Function Testing," the author makes the excellent point that "interpretation of PFTs is best made if the clinical question asked of the test is considered." He discusses several disease entities whose diagnosis and treatment depend on PFT. Unfortunately, he characterizes (in bold face type) asthma as primarily a disease of bronchoconstriction, and, further, says that bronchodilators are the first line drug, with steroids being used only in those patients who fail to respond to bronchodilators.

Chapter 2, "Spirometry and Related Tests," discusses standard spirometry and all measured or derived values, flow-volume loops, peak expiratory flow, maximum voluntary ventilation (MVV), pre- and post-bronchodilator studies, maximal inspiratory and expiratory pressures, airway resistance and conductance, and compliance. Three case studies go into great detail in explaining the results and their significance. Unfortunately, in this chapter, morbidity is defined as the "likelihood of dying," whereas the dictionary defines it as state of sickness.

Chapter 3 addresses "Lung Volumes and Gas Distribution Tests." Techniques for measuring lung volumes containing residual volume are discussed, using gas dilution techniques as well as body plethysmography.

Chapter 4, on "Ventilation and Ventilatory Control Tests," treats the assessment of response to carbon dioxide and oxygen. It is here that the editing starts to get in the way of the message. The volume of gas expired per minute is given the symbol V_E (with no dot over the V); then, an entire paragraph is repeated. In the self-assessment questions at the end of the chapter, the diagnosis of hypoventilation is determined by both an arterial carbon dioxide tension (P_{aCO_2}) of 49 mm Hg *and* a pH of 7.29. These missteps are jarring when one is reading and has to pause to discern whether the book is in error.

Chapter 5 covers "Diffusing Capacity Tests." More tests than I knew existed are covered in this chapter, with heavy emphasis on the single breath technique as the most commonly used.

Chapter 6, "Blood Gases and Related Tests," is especially frustrating, partly because of editing problems. In the first paragraph the author states that "Noninvasive techniques have drawbacks. Understanding of their limitations allows them to be used to provide appropriate patient care." Shortly thereafter, he mentions the units for blood gas measurement as torr or kPa, but provides normals only for torr. In describing blood gas sampling, the amount of heparin used in the syringe is described as "0.25 mL of 1000 μ/ml" (L is the symbol for liter and μ stands for "micro" as opposed to units). He does make the important point that the clinical importance of temperature correction is unclear (what should your P_{O_2} be if your temperature is 32° C?) and, thus, blood gases should be reported at 37° C. Further along, the pulmonary function technologist is encouraged to monitor ventilation-perfusion ratio ($\dot{V}/\dot{Q}$) alterations by "... allowing the subject to breath [sic]100% O_2 ...". Later, in the discussion of shunt calculation, he states that "approximately 5% of the cardiac output is shunted *through* the pulmonary system... ". There is a small shunt calculated for healthy people—it is a result of the bronchial and thebesian drainage into the left side of the heart. In that paragraph, the discussion addresses what happens when the "... terminal airways loose [sic] their support... ". At the end of this section, in the case in which $C_{(a-v)O_2}$ cannot be estimated or hemoglobin cannot be saturated with oxygen, he recommends using $P_{(A-a)O_2}$ as in index for $\dot{V}/\dot{Q}$ matching. There are 2 problems here. First, he uses the symbols A-aD_{O2} for the alveolar-arterial oxygen gradient; in this context, the D usually stands for delivery. Second, the $P_{(A-a)O_2}$ changes with F_{IO_2}; the only normals published are for someone breathing room air. At the end of the chapter, the first case study reports a D_L/V_A of 76, when predicted is 4.38. The second case study addresses hemoglobin concentration in the discussion, but no hemoglobin measurement was given in the presentation. The chapter summary includes the following example of the problem of subject/verb agreement: "The technical aspects of obtaining samples was covered...". Finally, Question 3 asks the result of having too much heparin in a blood gas sample. The answer given is lowering of P_{CO_2} because of dilution. Alteration of the P_{O_2} and lowering of pH are separate (incorrect) answers.

Chapter 7, "Cardiopulmonary Exercise Testing," discusses the differential diagnosis of the causes of dyspnea (cardiac, pulmonary, deconditioning, poor effort) as one major topic. Also included are oxygen consumption and carbon dioxide production, calculation and significance of respiratory exchange ratio, and changes in cardiorespiratory variables during exercise. In this chapter, the author recommends that the personnel be *certified* in cardiopulmonary resuscitation; people receive course completion cards, but the term *certification* has not been used for several years because of legal implications. Also, the author recommends that the emergency cart contain propanolol [sic]. There is some confusion apparent in the discussion of minute ventilation. The author defines $\dot{V}_{Emax}$ as "the $\dot{V}_E$ achieved at the highest work load attained." Actually, $\dot{V}_{Emax}$ is the maximum minute volume of which the subject is capable. The author then compounds the problem with descriptions of ventilatory reserve. In a normal subject, the limiting factor in O_2 delivery is the cardiovascular system. Thus, there normally is some ventilatory reserve at $\dot{V}_{O2max}$. This could be expressed as the difference between $\dot{V}_E$ at $\dot{V}_{O2max}$ and MVV (estimated as the forced expiratory volume in the first second [FEV_1] × 35) divided by MVV. Normally, the $\dot{V}_E$ at $\dot{V}_{O2max}$ is about 60–70% of MVV. In pulmonary disease, the lungs become the limiting factor in O_2 delivery. In this case, the $\dot{V}_E$ at $\dot{V}_{O2max}$ approaches the MVV; that is, the percentage approaches 100. Unfortunately, these 2 sentences completely misstate these facts: "In healthy subjects the ventilatory reserve seldom exceeds 50%. In subjects with pulmonary disease, it typically exceeds 70%." The rest of the chapter does well until the case studies. Here, smoking history is given in "pack/years" instead of pack-years. Also, in Case 7C, the arterial oxygen tension (P_{aO_2}) for a particular blood gas in the text is different from that in the table.

Chapter 8, "Specialized Test Regimens," covers methacholine inhalation challenge, histamine challenge, exercise challenge, eucapnic hyperventilation, preoperative PFT, and testing for disability. A little-known area is PFT in children; how do you measure pulmonary function when the subject cannot cooperate? The chapter concludes with indirect calorimetry. In this chapter, problems begin early on, when the author indicates that "Methacholine is a chemical stimuli...". Later, he incorrectly defines morbid obesity as "greater than 30% above ideal body weight" (in fact, obesity is generally defined as greater than 20% above ideal body weight and morbid obesity as greater than 100% above ideal body weight). Awkward phrasing continues in the discussion about performing PFTs in children: "The functional residual capacity in infants not only is a function of the recoil forces of the lung and chest wall but is dynamically maintained...". In the section on indirect calorimetry, we learn that the "... decrease in spirometer volume equals the rate of O_2 uptake of V_{O_2}." One of the questions at the end asks how a patient referred for disability determination should be evaluated. The indicated answer is 4–6 minutes of treadmill exercise at 5 METs, with blood gases. But because disability is relative to the person's job responsibilities and energy expended on the job, a standard recipe does not fit every situation.

Chapter 9,"Pulmonary Function Testing Equipment," covers all types of equipment, not only for formal pulmonary function testing, but also for peak flow measurement, blood gas analysis, gas analysis, and oximetry (by both transmission and reflectance spectrophotometry). This chapter is remarkably well written and edited, compared to the other chapters. It stumbles only on the question of what forms of hemoglobin are measured by pulse oximetry, its answers being RHb (reduced hemoglobin or that which is not carrying oxygen) and total Hb.

The last chapter addresses "Quality Assurance in the Pulmonary Function Laboratory," an important subject in that failure to utilize proper quality control procedures can have substantial effects on patient care. One quite interesting recommendation is the use of a lung analog to check the performance of DL_{COsb} (single-breath diffusion of carbon monoxide across the lung). This enables simultaneous check of the helium and carbon monoxide analyzers, volume transducers, breathing circuit, and software. The ratio of the 2 gases should be 1, and the calculated DL_{CO} should be 0.

The appendixes address reference values, conversion and correction factors, regulations and regulatory agencies, equations, sample calculations, and the mean and standard deviation. Unfortunately, the problems with the text persist into the appendixes. The appendix on conversion and correction factors defines work, power, and energy in Joules (energy should be in joules per second). In the same chart, the SI unit for area is 0 instead of m^2; L/kPa is written as L/k/ Pa. The appendix on regulations and agencies discusses the Joint *Committee* on Accreditation of Health care Organizations, mentions the American Review of Respiratory Disease (which changed its name at least 5 years ago), and the chart has some typesetting problems that make it difficult to figure out some sections. In the appendix on equations, the author states: "If all pulmonary capillary blood equilibrates with alveolar gas, C_{cO_2} and C_{aO_2} become identical. . .". As blood from the bronchial circulation and from the coronary circulation drains into the left side of the heart, C_{aO_2} must *always* be less than C_{cO_2}, and calculated shunt will always be greater than zero.

The glossary is riddled with inaccurate or confusing definitions, some of which have no direct relationship to the topic of the book. For example, anatomic dead space is said to go "down to the level of the respiratory bronchioles." Since the respiratory bronchioles are, by definition, part of the gas exchange area, they cannot be part of the anatomic dead space. A buffer is defined as "a solution that will maintain a given pH. . . ," but the pH has to change some for the buffer to function. The pH will not return to its original value, but the change will be less than if there were no buffer present. Ejection fraction is defined as "the portion of ventricular volume ejected during systole." Specifically, it is the ratio of stroke volume to end diastolic volume. The definition provided for rebreathing is incomprehensible: "referring to or describing a system which allows the subject to breathe continuously while measurements are made."

The present review includes only some of the problems I identified in reading the **Manual of Pulmonary Function Testing**, and it concerns me that respiratory therapists in training might obtain and memorize a significant amount of incorrect information or be left confused by some parts of the book. I would encourage the author and editor to make a meticulous review prior to the next edition.

Robert R Fluck Jr RRT
Department of Cardiorespiratory Sciences
SUNY Health Science Center
Syracuse, New York

Exam Review and Study Guide for Perinatal/Pediatric Respiratory Care, Robert Harwood MSA RRT. Soft-cover, illustrated, 350 pages. Philadelphia: FA Davis Company, 1999. $32.95.

This book is addressed to several audiences: candidates for the National Board of Respiratory Care (NBRC) Perinatal/Pediatric Specialty Exam; students in respiratory care training programs; and entry level practitioners in the field of perinatal/pediatric respiratory care. Toward the needs of the first group, there are two 120-question pretests and post-tests, patterned after the NBRC's Detailed Content Outline (which governs construction of the specialty exam) and detailed analysis of the answers.

Each of the 16 chapters begins with an outline of the chapter and end with a self-test. The self-tests include open-ended and multiple-choice questions. All the chapters reference very recent publications, and some reference materials on the World Wide Web.

Harwood maintains a consistent style, approach, depth, and quality. For instance, most chapters provide considerable detail on nonpulmonary aspects of pediatric pathophysiology and care. Especially prominent is an appreciation of the role of the patient's level of development in the therapist's planning and delivery of care.

The book is illustrated with simple black and white line drawings, mostly of good quality. No photos are included in the illustrations, presumably in order to keep the cost of the book low. The first chapter consists of an overview of the NBRC's Perinatal/Pediatric Specialty exam, as well as test-taking and study tips. The remaining chapters are clinically oriented. Chapter 2, "Developmental Care of the Newborn," begins with a good introduction to the ideas of Dr Heidelise Als, which have transformed neonatal intensive care during the last decade. Chapter 3 reviews fetal lung development.

Chapter 4 covers comprehensive assessment of the fetus and newborn. In addition to cardiopulmonary assessment, Harwood discusses topics such as glucose and electrolyte abnormalities, which, unfortunately, are not always covered in respiratory care texts. The otherwise excellent discussion of chest radiograph interpretation is flawed by a lack of illustrations.

Chapter 5 addresses newborn resuscitation. Chapter 6 provides an overview of common respiratory and nonrespiratory diseases, including sepsis and intraventricular hemorrhage. Chapter 7 presents all aspects of monitoring in the newborn and child, and briefly discusses pediatric CPR (no reason given why this section was not included in Chapter 5). Chapter 8 discusses pediatric assessment, and, like the neonatal chapter, includes nonrespiratory issues as well, though not to the same degree.

Chapter 9 covers oxygen and humidity therapy, and probably includes too much information on the basics of these therapies, which pediatric specialists would presumably know from their general training. The discussion of retinopathy of prematurity is generally excellent, but mistakenly refers to laser therapy to treat glaucoma in retinopathy of prematurity (lasers are actually used to treat or prevent vascular proliferation and retinal detachment). The discussion of humidifier/closed incubator interactions is succinct and well illustrated. However, heated humidifier/radiant warmer interactions are not discussed.

Harwood also mistakenly advocates the use of weight-based or age-based formulas to calculate the dose of aerosolized drugs. The fact that the infant's and child's minute ventilation (and therefore, drug inhalation) is proportional to weight obviates the need to deliver smaller doses of aerosolized drugs to these patients. The discussion of continuous nebulization of ribavirin and other agents in Chapter 9 is inadequate, given the widespread practice of nebulization during mechanical ventilation. Furthermore, some recommendations, such as placing the nebulizer between the Y adapter and the endotracheal tube have been shown to reduce aerosol deposition. Chapter 9 also includes a discussion of continuous positive airway pressure and bi-level positive airway pressure. In future editions of this book the chapter's title should be changed to better reflect its contents.

Chapter 10 covers the most common pediatric pulmonary disorders. Included is the seemingly obligatory "croup versus epiglottitis" table, which has appeared in almost every pediatric respiratory or critical care text published during the last 30 years. In that the *Haemophilus influenzae* type B vaccine has virtually eliminated epiglottitis in the USA and other developed countries, perhaps it is time to eliminate the epiglottitis discussion as a standard feature of pediatric texts.

The section on pediatric asthma is one of the most thorough and accessible in recently published respiratory texts, and fully reflects the most recent recommendations of the National Asthma Education Project. Likewise, the review of cystic fibrosis does a good job of explaining the function of the cystic fibrosis transmembrane regulator (the defective gene that causes cystic fibrosis). The discussion of cystic fibrosis could be improved by a discussion of the role of *Burkholderia cepacia* in the pathophysiology of cystic fibrosis in older children and young adults, as well as its impact on the management of cystic fibrosis centers. The book would also benefit from discussions on gastroesophageal reflux and respiratory disorders in the immunocompromised child.

Airway management and postural drainage are discussed in Chapter 11. An overview of pediatric CPR and advanced life support is included, but is not listed in the index. Also missing is a critical review of recent "hot topics" in the pediatric nursing and respiratory literature, such as the routine use of normal saline during suctioning, suctioning frequency, depth of catheter insertion, and the best means of maintaining oxygenation and asepsis during suctioning.

Chapter 12 covers congenital heart defects, and each of the major defects is illustrated. Unfortunately, the discussion of surgical repair is superficial. It would be worthwhile to include a discussion of common issues in postoperative care of the pediatric heart patient, such as management of pulmonary hypertension. Harwood briefly mentions the use of innovative therapies such as inhaled CO_2, but fails to describe the rationale or technique.

Chapter 13 thoroughly discusses mechanical ventilation and includes several useful tables. Chapter 14 features a well illustrated discussion of the use of pulmonary function testing in the ventilated patient. Most of the illustrations are from commercial sources. Chapter 15 discusses special procedures such as chest tube insertion and extracorporeal membrane oxygenation. The final chapter is on home care, with a special emphasis on apnea monitoring.

Despite its relatively low price, this text meets its goal of providing a comprehensive introduction and review of perinatal and pediatric respiratory care.

Robert Lewis ND RRT
Family Nurse Practitioner
Healthsouth
Seattle, Washington

Management Skills for the New Health Care Supervisor, 3rd ed, William Umiker MD. Hardcover, 423 pages. Gaithersberg MD: Aspen Publishers; 1998. $49.00.

A preponderance of health care workers promoted to supervisory positions find themselves embarking on their new career without the benefit of managerial training or experience. They have reached this position because they are technically astute, but find themselves in unfamiliar territory and are expected to supervise the same people who were formerly co-workers. This involves numerous new experiences and a whole new set of problems. Umiker's book aims to smooth the transition from worker to middle manager. It is a textbook of how to deal with the day-to-day "people problems" that consume a great deal of a new supervisor's time and energy. The content is appropriate for therapists, nurses, physicians, and other health care workers who move into management positions. The organization of the book follows a natural progression from "I've got the job and now what do I do with it?" to managing people and developing communication skills, and even touches on the dreaded topic of cost control and managed care.

Part I, "Planning and Organizing," details how radically work life can change during the leap from "doing" skills to "leading" skills. It describes the skills needed, the changes in the various work relationships (former co-workers now subordinates, other supervisors, one's new manager), and identifies potential pitfalls. Customer service, planning, organizing, orientation of new employees, and team-building are also discussed in this section.

Part II, "Controlling People," focuses on managing staff behavior, both positive and negative. Cultural diversity, performance feedback, and employee retention are discussed herein. Most of us will be able to identify one or two of our own employees profiled in the behavior type sections.

Part III, "Cost Control," briefly addresses key concepts of cost control, budgets, and managed care. The author clearly states that this is not the focus of the book, but only a quick overview of the essentials. Even though supervisors are not often solely responsible for the budget, their job frequently requires input on the subject, and this section is certainly beneficial.

Parts IV and V, "Developing Employees" and "Special Supervisory Skills," detail the specific skills needed to encourage creativity, enhance verbal and written communication, and improve negotiation skills and time management. Part VI discusses coping with stress, career development, planning for a successor, and the importance of networking with fellow supervisors.

Each chapter lists references and recommended reading. Some of the quotes and sayings interspersed among the chapters are appropriate and insightful, while others are somewhat trite, but they do lighten what might otherwise be very dry material. There is no need for illustrations in the book, but the use of tables and bold headings helps make information easy to find.

In summary, this book is well written, organized, and indexed, contains a wealth of pertinent information for the new supervisor, and is well worth reading before embarking on a new management position. It would also benefit experienced supervisors seeking fresh or renewed perspective, or greater efficiency in their current position.

Carol L Kasper RRT
Department of Respiratory Care
Harborview Medical Center
Seattle, Washington

High Life: A History of High-Altitude Physiology and Medicine, John B West PhD MD DSc. Hardcover, illustrated, 493 pages. Oxford: Oxford University Press for the American Physiological Society; 1998. $79.50.

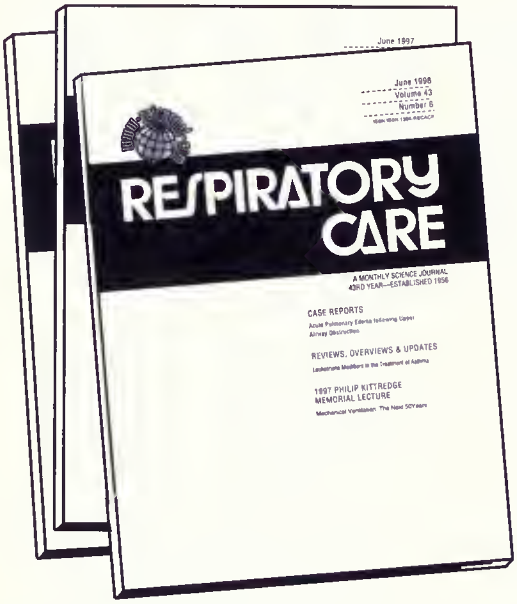
High Life is an extensive history of high altitude physiology, intended for a broad audience of biologists, physiologists, physicians, and those interested in trekking and climbing at high altitude. John West has a long involvement with pulmonary physiology research, including high altitude studies. He was the leader of the 1981 American Medical Research Expedition to Mt Everest, which was successful from both climbing and scientific perspectives. As with other books by John West, this one is well written and very thorough, so much so that I was tempted to skim some parts where controversial historical points were discussed in detail.

After briefly discussing what the ancient Greeks and Chinese wrote about high altitude's effects on the body, the first 4 chapters chronicle discoveries in atmospheric science and physical chemistry relating to barometric pressure and altitude. This discussion includes an excellent summary of the early history of pulmonary physiology in the 17th to 19th centuries, and the related early history of high altitude medicine. Early in the book West also discusses in detail the observations made by Father Joseph de Acosta on the effects of altitude on the human body. Father de Acosta, a Jesuit priest living in South America in the 16th century, is generally credited with the first description of high altitude illness. West's history of pulmonary physiology should be interesting to anyone who works in a field related to lung biology or lung disease.

The middle of the book includes a chapter on the history of high altitude illness, another on early expeditions to Mt Everest, and another on permanent residents of high altitudes. Devoting a whole chapter to discussing the people involved in first recognizing high altitude illness helps to give credit to some earlier descriptions of acute mountain sickness and high altitude pulmonary edema that are not often mentioned in the literature. This might be of most interest to persons involved in high altitude illness research. The chapter on early expeditions to Mt Everest, however, would be of interest to most readers, because the history of early Mt Everest attempts sets the stage for the physiologic extreme of living and climbing above 20,000 ft. Subsequent research projects described later in the book are Operation Everest I and II, in which subjects were taken to an equivalent barometric pressure as that at the summit of Mt Everest, but over the duration of several weeks and in a chamber. Other field expeditions include the Silver Hut Expedition in the Everest region of Nepal, and the American Medical Research Expedition to Everest. We see that the environment and climbing history of Everest are intimately related to the history of modern high altitude physiology research.

Later chapters describe the more recent research on high altitude illness and physiology, providing a comprehensive modern day understanding. Overall this book offers a fascinating look at the history of high altitude physiology, and an appreciation of our current understandings. The only criticism I might make is that West is so thorough in some aspects of his endeavor to describe the history of high altitude physiology that parts of the book are slower reading. I am sure, however, that what was not as interesting to me will be fascinating to a reader with a different background. This really speaks to the variety of information in the book. Most everyone interested in pulmonary physiology or high altitude illness will find at least some part of this book fascinating reading.

Colin K Grissom MD
Pulmonary Division
LDS Hospital
Salt Lake City, Utah


June 1997
June 1998
Volume 43
Number 6
RESPIRATORY CARE
A MONTHLY SCIENCE JOURNAL
43RD YEAR—ESTABLISHED 1956
CASE REPORTS
REVIEWS, OVERVIEWS & UPDATES
1997 PHILIP KITTREDGE MEMORIAL LECTURE

CRCE through the Journal—1999

CRCE through the Journal, a program for American Association for Respiratory Care (AARC) members to gain credit for continuing education, is now in its eleventh year. By reading RESPIRATORY CARE—the science journal for respiratory care professionals—and completing this examination, AARC members may earn credit for continuing education.*

This 50-item, multiple-choice examination is based on papers published from July 1998 through June 1999 in RESPIRATORY CARE. The issue and page numbers of the paper on which a question is based are shown in brackets following the question. You may consult the cited paper; however, we *encourage* you to read the paper in its entirety before answering the question. Choose the single most-correct answer, and mark the answer sheet, which is located following Page 992.

Mail your completed answer sheet by September 30, 1999. Answer sheets postmarked after September 30 will not be processed. The Answer Key for ***CRCE through the Journal*** will be published in the October Issue of RESPIRATORY CARE. No scores will be available from the AARC until the 1999 CRCE Transcripts are mailed in early 2000.

We are indebted to Phillip Hoberty EdD RRT, Assistant Professor and Director of Clinical Education, Respiratory Therapy Division, School of Allied Medical Professions, The Ohio State University, Columbus, Ohio; Mike Tissue MS RCIS RCS RPFT RRT Perinatal/Pediatric Specialist, Cardiopulmonary Care Sciences, Georgia State University, Atlanta, Georgia; Timothy B Opt'Holt EdD RRT and William V Wojciechowski MS RRT, Department of Cardiorespiratory Care, University of South Alabama, Mobile, Alabama; and Linda L Stone RRT, Program Director, Northern Virginia Community College, Annandale, Virginia.

---

*The acceptance of these credits for the fulfillment of license-mandated continuing education is dictated solely by the licensure law of each individual state.

QUESTIONS:

Please follow the instructions on the previous page, and record your answers on the perforated form provided following Page 992.

1. According to Stoller and Michnicki, which of the following are noteworthy shortcomings of their research?
   - a. The possibility that respondents comprised a biased sample.
   - b. The recorded perceptions were not representative of all medical house officers.
   - c. The possibility that respondents were inclined to provide favorable answers.
   - d. The surveyors did not assess the house officers expertise in ordering respiratory care services.
   - e. All of the above.

   [July 1998;43(7):549-551]

2. Hussey et al reported that respiratory therapists trained to use a decision tree algorithm intubated
   - a. 104 patients, unsupervised by Emergency Department physicians.
   - b. 54 patients, unsupervised by anesthesiologists.
   - c. 162 patients, supervised by anesthesiologists.
   - d. 75 patients, supervised by anesthesiologists.
   - e. 89 patients, supervised by Emergency Department physicians.

   [July 1998;43(7):552-556]

3. According to Gupta et al, 63 men were seen in the Emergency Services Department 48 hours following exposure to toxic fumes from an accidental underground explosion; some of the men presented with
   - a. no symptoms.
   - b. dyspnea, chest discomfort, and vomiting.
   - c. burning in the eyes, headache, and giddiness.
   - d. severe respiratory distress.
   - e. all of the above.

   [July 1998;43(7):557-561]

4. According to Ludlam et al, which of the following have NOT been noted as rare causes of endobronchial lesions in some HIV-infected patients?
   - a. *Pneumocystis carinii*
   - b. Bacterial and cytomegalovirus tracheitis
   - c. *Escheria coli*
   - d. Grandular cell myoblastoma
   - e. *Rhodococcus equi*

   [July 1998;43(7):562-566]

5. According to Hill, what trend has occurred during the past dozen years or so in the care of ventilator-assisted individuals (VAIs)?
   - a. Acute care hospitals established on-site ventilator units.
   - b. Subacute and chronic care hospitals failed to open weaning units.
   - c. The total number of VAIs in the population has decreased.
   - d. Noninvasive ventilation, particularly NPPV, has steadily declined in use.
   - e. A national database has been established to track the care of VAIs.

   [August 1998;43(8):635-636]

6. What result was found when Stoller et al studied patterns of adult non-ICU respiratory care services at The Cleveland Clinic Foundation while using a respiratory therapy consult service (RTCS)?
   - a. The total number of high-volume therapies increased during the 5-year period.
   - b. The proportion of bronchodilator therapy delivered by small volume nebulizer increased.
   - c. The number of patient days for oxygen therapy decreased with a resulting savings of several thousand dollars.
   - d. It was shown that the use of the RTCS caused a decrease in therapy volume and therapy cost.
   - e. The hospital census steadily declined during the study.

   [August 1998;43(8):637-642]

7. What did Adams et al find to be true when they looked for changing prevalence and characteristics of ventilator-assisted individuals (VAIs) in Minnesota in 3 cross-sectional surveys?

   a. The number of VAIs increased from 1986-1997 in the same proportion as the total population of the state.
   b. Chronic obstructive pulmonary disease was the predominant reason for ventilator assistance.
   c. Inclusion of NPPV in the third survey indicated it contributed to nearly half of the increase in ventilator assistance from 1992 to 1997.
   d. The most prevalent age group for ventilator assistance was 31-40 years of age.
   e. The inclusion of all VAIs in the state was assured.

   [August 1998;43(8):643-649]

8. According to Salyer, what is needed to support routine continuous pulse oximetry monitoring?

   a. proven effect of monitoring on patient outcomes
   b. further studies on the accuracy of pulse oximetry
   c. evidence of widespread use of pulse oximetry in the medical-surgical patient population
   d. assumption that it is the "standard of care" and cannot be abandoned
   e. evidence that it is commonly used in ICU settings

   [August 1998;43(8):650-654]

9. What statement is consistent with the AARC Position Statement: Administration of Sedative and Analgesic Medications by Respiratory Care Practitioners?

   a. The AARC believes that RCPs should not, even under direct physician supervision, participate in administering prescribed sedation and analgesia.
   b. The American Society of Anesthesiologists offers no published guidelines on the non-anesthesiologist offering sedation and analgesia.
   c. After training and competency assessment, RCPs should be certified as competent by the appropriate medical director and department head or governing body in their facility before offering these services.
   d. The AARC Position statement applies to diagnostic, but not to therapeutic, procedures.
   e. JCAHO requirements and policies do not apply to offering these services.

   [August 1998;43(8):655]

10. Which statement is true about Rau et al's study of in-line MDI actuators for delivery of aerosolized drugs?

    a. Only the delivery of β-agonist drugs were studied.
    b. The purpose of the study was to evaluate dose delivery through the oral route.
    c. The study used a mechanically ventilated dog lung model.
    d. The results were the same for all 5 devices tested.
    e. In vitro results reported support the need for an in vivo clinical comparison.

    [September 1998;43(9):705-712]

11. What statement is consistent with Stoller's review of research on use of protocols in respiratory care?

    a. Misallocation of respiratory care services occurs commonly in patient care.
    b. Use of respiratory-care-implemented protocols decreases misallocation of services, but increases cost and frequency of adverse clinical outcomes.
    c. The few carefully controlled, randomized trials of protocols that have been performed fail to support the use of protocols.
    d. The effects of using respiratory care protocols should be confined to the acute care setting.
    e. The definition of misallocation of care is restricted to "over-ordering of respiratory care."

    [September 1998;43(9):719-723]

12. Based on the Point of View by Kacmarek and Chiche about ventilator treatment of patients with acute respiratory distress syndrome (ARDS), what approach to ventilator care is appropriate?

    a. Ventilator strategies in ARDS should routinely encourage high peak ventilating pressures.
    b. PEEP should be avoided early in the ventilator treatment of ARDS.
    c. A lung protective ventilator strategy (LPVS) is generally not beneficial.
    d. Maximal distention of the alveoli with high tidal volumes is useful in avoiding complications in ARDS.
    e. High PEEP levels may be necessary when based on the actual respiratory mechanics of the individual patient.
    [September 1998;43(9):724-727]

13. The results of Aslamy et al's study of cardiac enzyme as evidence of cardiac injury in intubated patients with acute respiratory failure (ARF) encouraged what approach to assessing these patients?

    a. Use creatine kinase (CK) screening in patients with pulse rate >140 bpm or history of coronary artery disease.
    b. Perform enzyme screening of all ventilated patients >45 years old with noncardiac respiratory failure.
    c. Assume that intubated patients in noncardiac respiratory failure do not experience cardiac injury.
    d. Assume that isolated, elevated cardiac enzymes in ARF predict altered clinical outcomes.
    e. Use chest pain and ECG changes as the only approaches to selecting intubated patients for enzyme screening.
    [September 1998;43(9):713-718]

14. Which of the following was encouraged in the Classic Reprint, "Standards of Effective Administration of Inhalation Therapy" (1950)?

    a. delegating inhalation therapy services to the intern staff, nurses, and orderlies
    b. registration of technicians and technician supervisors by written and oral examination
    c. excluding a physician outside of anesthesiology from serving as medical director for inhalation therapy services
    d. eliminating the teaching of inhalation therapy in medical schools and hospitals
    e. implementing a formal accrediting body for schools of inhalation therapy
    [September 1998;43(9):730-748]

15. Based on the program evaluation findings presented by Powell, low knowledge and confidence about asthma management by school personnel improved after 6 months when they received which of the following sessions of instructions?

    a. 30 minute class on asthma management
    b. 1 hour class on asthma management
    c. 2 or 3 hour class on asthma management
    d. 4 hour class on asthma management
    e. 8 hour class on asthma management
    [October 1998;43(10):804-810]

16. Which of the following is NOT an opioid analgesic?

    a. ibuprofen
    b. morphine
    c. fentanyl
    d. codeine
    e. methadone
    [November 1998;43(11):942-951]

17. The data reported by Arnold suggest that early institution of high-frequency oscillatory ventilation (HFOV) in pediatric patients with acute lung injury is associated with which of the following?

    a. slightly higher incidence of subsequent chronic lung disease
    b. moderately higher incidence of subsequent chronic lung disease
    c. considerably higher incidence of subsequent chronic lung disease
    d. lower incidence of subsequent chronic lung disease
    e. no difference in the incidence of subsequent chronic lung disease
    [November 1998;43(11):961-965]

18. The predominant criterion, according to Dalton and Heulitt, for placing a pediatric patient on extracorporeal membrane oxygenation (ECMO) remains as:

    a. aspiration.
    b. bacterial pneumonia.
    c. viral pneumonia.
    d. myocarditis.
    e. failure to respond.
    [November 1998;43(11):966-977]

19. According to Dalton, the by-product of inhaled nitric oxide (iNO) and oxygen yields nitrogen dioxide (NO_2), a more highly toxic chemical that produces damage when inhaled in amounts as low as _____ parts per million.

    a. 5
    b. 10
    c. 15
    d. 20
    e. 25
    [November 1998;43(11):978-987]

20. In the Schexnayder and Heulitt study, which factor was reported by most participating practitioners (46%) as the one they believed to be the most important in preventing lung damage during mechanical ventilation?

    a. peak inspiratory pressure
    b. tidal volume
    c. oxygen concentration
    d. positive end-expiratory pressure
    e. arterial carbon dioxide tension
    [November 1998;43(11):995-998]

21. Although the American Thoracic Society has not yet adopted guidelines for acceptability of the inspiratory flow-volume (FV) curve, Hnatiuk et al state which of the following as a useful criterion of acceptability?

    a. $FIF_{50} \geq 95\%$ MIF
    b. $FIF_{50} \geq 90\%$ MIF
    c. FIVC ≥ FVC
    d. FIVC ≥ 90% FVC
    e. parabolic shape with only one inflection point
    [December 1998;43(12):1058-1063]

22. Which mode of ventilation is preferred by Schinco et al for bronchopleural fistual management?

    a. volume control
    b. SIMV
    c. pressure control
    d. pressure support
    e. CPAP
    [December 1998;43(12):1064-1069]

23. Which of the following statements is true regarding pulmonary surfactant?

    a. It functions to increase surface tension at all lung volumes and decreases surface tension as alveolar surface decreases.
    b. It functions to increase surface tension at all lung volumes and increases surface tension as alveolar surface decreases.
    c. It functions to lower surface tension at all lung volumes and increases surface tension as alveolar surface increases.
    d. It functions to lower surface tension at all lung volumes and increase surface tension as alveolar surface decreases.
    e. It functions to lower surface tension at all lung volumes and decreases surface tension as alveolar surface decreases.
    [December 1998;43(12):1070-1085]

24. According to Sibbald, in addition to measuring outcomes, which of the following is NOT a core competency in the health services domain that he feels our system needs at this time?

    a. risk stratification
    b. nutritional approaches
    c. assessment of cost effectiveness
    d. measurement of the quality of life and the quality of care
    e. measurement of the quality of life and the quality of care economic evaluations
    [December 1998;43(12):1092-1098]

25. According to Mitchell et al, which of the following previous studies was most similar to their study of the effects of metered-dose-inhaler propellant on holding chamber performance?

a. Dolovich, 1993
b. Barry and O'Callaghan, 1995
c. Barry and O'Callaghan, 1997
d. Wildhaver et al, 1998
e. Schulz et al, 1996
[January 1999;44(1):38-44]

26. Analogs of which of the following molecules competitively inhibit the action of nitric oxide synthase (NOS)?

a. guanylyl cyclase
b. L-arginine
c. protein kinase
d. cyclic guanidine monophosphate
e. citrulline
[February 1999;44(2):156-166]

27. Which of the following cytotoxic effects are related to the inhalation of nitric oxide?

I. DNA damage
II. increased aerobic metabolism
III. inhibition of mitochondrial enzymes
IV. stimulation of RNA replication

a. II, III only
b. I, III only
c. II, IV only
d. I, III, IV only
e. I, II, III, IV
[February 1999;44(2):156-166]

28. Which of the following physiologic effects are associated with the inhalation of nitric oxide?

I. decreased platelet aggregation
II. inhibition of smooth muscle cell proliferation
III. decreased intracranial pressure
IV. decreased inflammatory response

a. I, II only
b. III, IV only
c. II, III, IV only
d. I, II, IV only
e. I, II, III, IV
[February 1999;44(2):212-219]

29. Which types of gas analyzers are suitable for monitoring nitric oxide and nitrogen dioxide in breathing systems?

I. infrared absorption analyzers
II. chemiluminescence analyzers
III. emission spectroscopy analyzers
IV. electrochemical analyzers

a. I, IV only
b. II, IV only
c. II, III only
d. I, II only
e. I, II, III only
[March 1999;44(3):281-306]

30. Which of the following descriptions characterize nitric oxide gas?

I. Nitric oxide is an unstable, free radical in atmospheric air.
II. Nitric oxide reacts slowly with oxygen to form nitrogen dioxide.
III. Nitric oxide is a highly lipid soluble.
IV. Nitric oxide reacts with water to form nitric acid.

a. I, III only
b. I, IV only
c. I, II, III only
d. II, III, IV only
e. I, II, III, IV
[March 1999;44(3):281-306]

31. Which of the following statements refer to inducible nitric oxide synthase (iNOS)?

I. It is located in endothelial cells.
II. It is calcium/calmodulin independent.
III. Its production is stimulated by endotoxin of Gram-negative bacteria.
IV. It produces small amounts of NO for short time periods.

a. II, III only
b. I, III, IV only
c. I, II, IV only
d. I, II, III only
e. I, II, III, IV
[March 1999;44(3):308-313]

32. Which of the following conditions are possible toxic effects of inhaled nitric oxide?

I. methemoglobinemia
II. nitrogen dioxide production
III. peroxynitrite production
IV. increased left ventricular filling pressure

a. II, III only
b. I, IV only
c. I, II, III only
d. II, III, IV only
e. I, II, III, IV
[March 1999;44(3):315-329]

33. Which of the following mechanisms possibly limit the NO effect through a defect of the NO-cyclic guanylate cyclase monophosphate signal transduction pathway?

I. decreased guanylate cyclase activity
II. increased phosphodiesterase activity
III. abnormal cGMP-dependent protein kinase
IV. impaired vascular smooth muscle contractility

a. I, II only
b. I, III only
c. II, IV only
d. II, III, IV only
e. I, II, III, IV
[March 1999;44(3):331-337]

34. Which of the following statements describe the interaction between inhaled nitric oxide and red blood cells?

I. Hemoglobin has an affinity of nitric oxide 10,000 times greater than it has for oxygen.
II. Fetal hemoglobin has less of an affinity for nitric oxide than adult hemoglobin.
III. Globin becomes oxidized when nitric oxide combines with hemoglobin.
IV. Ferrous heme converts to ferric heme when nitric oxide combines with hemoglobin.

a. I, III only
b. I, IV only
c. II, III only
d. II, IV only
e. I, II, III only
[March 1999;44(3):340-347]

35. Which of the following statements are true concerning exhaled nitric oxide?

a. The main source of exhaled nitric oxide is the lower respiratory tract.
b. Nasotracheally intubated patients have high exhaled nitric oxide levels.
c. The main source of exhaled nitric oxide is the upper respiratory tract.
d. Orotracheally intubated patients have high exhaled nitric oxide levels.
e. Equal amounts of nitric oxide come from the upper and lower respiratory tract.
[March 1999;44(3):349-357]

36. What factor reported by Kakizaki et al was most likely responsible for the improvement in chest wall mobility after respiratory muscle stretch gymnastics?

a. normal progression of time
b. improvement in the patient's disease state
c. improvement in FEV_1
d. decreased chest wall elastance
e. increased respiratory muscle force
[April 1999;44(4):409-414]

37. What result was reached by Clini et al regarding chronic tracheostomy in patients following an acute exacerbation of COPD that required mechanical ventilation?

a. improved short-term survival in those patients with chronic tracheostomy
b. a higher rate of exacerbation requiring antibiotics with chronic tracheostomy
c. a decreased tidal volume with chronic tracheostomy
d. a decreased maximum inspiratory pressure with chronic tracheostomy
e. an increased P_{aCO_2} with chronic tracheostomy
[April 1999;44(4):415-420]

38. Based upon the conclusions of Patel et al, what procedure should the respiratory therapist follow once a mechanically ventilated patient has been weaned to 5 cm H_2O PSV +5 cm H_2O CPAP?

a. Provide an additional 24 hours of PSV + CPAP to assure readiness for ventilator withdrawal.
b. Extubate the patient.
c. Provide an additional 24 hours of CPAP using the ventilator to assure readiness for ventilator withdrawal.
d. Provide an additional day of ventilation in the assist-control mode.
e. Extubate the patient and provide an additional day of mask CPAP.
[April 1999;44(4):421-427]

39. According to Delgado et al, what is NOT one of the purposes of tracheal gas insufflation?

a. aid in the reduction of alveolar distending pressures
b. improve efficiency of CO_2 elimination
c. potentially reduce ventilatory requirements
d. improve distal gas mixing due to turbulence
e. allow a decrease in the F_{IO_2}
[April 1999;44(4):428-433]

40. According to Delgado et al, what intervention is necessary during pressure controlled ventilation with continuous tracheal gas insufflation to maintain minute ventilation?

a. increase the inspiratory pressure
b. decrease the PEEP
c. insert a pressure-relief valve
d. increase the I:E
e. decrease the inspiratory time
[April 1999;44(4):428-433]

41. In a study of the effects of a pediatric asthma protocol, Lierl et al concluded that in the protocol group compared to the control group:

a. length of stay was significantly decreased.
b. unnecessary treatments were virtually eliminated.
c. the number of aerosol treatments decreased significantly.
d. the presence of concurrent oxygen therapy increased the number of treatments.
e. the length of stay until aerosols could be decreased to Q6 hours was unchanged.
[May 1999;44(5):497-505]

42. According to the oxygen protocol study by Konschak, the cost savings was based upon:

a. the replacement cost of nasal cannulas every day.
b. the cost of the unnecessarily used oxygen.
c. the therapist's time.
d. the cost of the pulse oximeters.
e. all of the above.
[May 1999;44(5):506-511

43. According to the oxygen protocol study by Konschak et al, the median number of dollars wasted per patient on unnecessary oxygen was:

a. $0.75.
b. $1.15.
c. $3.95.
d. $4.23.
e. $5.65.
[May 1999;44(5):506-511]

44. In the study of an audit system for a respiratory care protocol service, Kester and Stoller discovered an inconsistency when scoring the "rule out pneumonia" diagnosis. How was this category clarified in order to decrease this inconsistency?

a. Therapist evaluators were instructed to consider pneumonia if the patient was febrile.
b. Therapist evaluators were instructed to consider pneumonia if the patient had an elevated white blood cell count.
c. Therapist evaluators were instructed to consider pneumonia if the patient had purulent sputum.
d. Therapist evaluators were instructed to consider pneumonia if the patient had an infiltrate and decrease in oxygenation.
e. Therapist evaluators were instructed to assume the diagnosis of pneumonia based upon the written diagnosis of "rule out pneumonia."
[May 1999;44(5):512-519]

45. According to Waugh and DeKler, a significant change in the F_{DO_2} of a noninvasive positive pressure ventilator occurs when which of the following changes are made?

    a. the location of the point at which oxygen is added to the inspiratory circuit
    b. altering the inspiratory time
    c. changing the EPAP level, while maintaining the constant pressure gradient
    d. altering the oxygen flow
    e. all of the above
    [May 1999;44(5):520-523]

46. Hurford notes that in intubation situations in which the patient is lying on the ground or floor there is a higher rate of success when the operator is in which position?

    a. left lateral decubitus position
    b. kneeling
    c. straddling the patient
    d. right lateral decubitus position
    e. Sellick position
    [June 1999;44(3):615-626]

47. According to Branson, passive humidification devices are passive in operation only; the decision to use one requires active consideration of all of the following except

    a. body temperature.
    b. minute ventilatory requirements.
    c. presence of pre-existing pulmonary disease.
    d. presence of a bronchopleural fistula.
    e. pediatric application.
    [June 1999;44(3):630-641]

48. What size of cuffed endotracheal tube does Kline recommend for a 6-year-old child?

    a. 5.5
    b. 6.0
    c. 3.5
    d. 4.5
    e. 4.0
    [June 1999;44(3):650-658]

49. A bronchial blocker is a device that

    a. has application limited to right lung procedures.
    b. is an excellent alternative to a double lumen endotracheal tube when presented with a difficult airway.
    c. can be inserted without FOB.
    d. is designed for use with laser procedures.
    e. has a design flow prohibiting suction application.
    [June 1999;44(3):661-682]

50. Ritz states that the most significant and sensitive outcome assessment for heliox therapy is

    a. monitoring P_{ETCO_2} levels.
    b. a 5 mm Hg decrease in pulsus paradoxus.
    c. vocal quality change to high-pitched, cartoon-like.
    d. improvement in dyspnea.
    e. ability to delay intubation.
    [June 1999;44(3):686-689]

Make sure to double check your work and to mark the answer sheet clearly. Mail your completed answer sheet by September 30, 1999 to

CRCE through the Journal
11030 Ables Lane
Dallas TX 75229-4593

Look for the answers in the October 1999 issue of RESPIRATORY CARE.

INTUBATION RESOURCES FROM THE AARC

IISPs

Tracheal Intubation I: Upper Airway Anatomy and Goals of Intubation
After completing this IISP, you will understand the rationale for tracheal intubation and be able to identify the important landmarks of upper airway anatomy.
Item CP4 $12 (nonmembers $16)

Tracheal Intubation II: Routes of Intubation
Describes the four routes of tracheal intubation and some advantages and hazards of each. Also presents the process for selecting the most suitable route in a given situation.
Item CP5 $12 (nonmember $16)

Equipment Procedures for Intubation
Identifies all equipment required to perform either oral or nasal tracheal intubation, while explaining necessary steps before and after intubation. Discusses common errors and differences in procedures for infants and pediatric patients.
Item CP6 $12 (nonmember $16)

VIDEOTAPES

Airway Management: Tricks of the Trade
Learn how to manage the obstructed airway and how to determine when an artificial airway is necessary. Also presented are the methods of assessing endotracheal tube positioning, monitoring the intubated patient, and determining when extubation is feasible. Featuring Charles G. Durbin, Jr., MD, FCCM, and David J. Pierson, MD.
Item VC67 $49.95 (nonmember $99)

Application of Positive Airway Pressure Without Intubation
Covers short-term application in the inpatient setting in the treatment of acute, life-threatening conditions and elective, long-term application in home care. Includes a discussion of bi-level positive airway pressure via the BiPAP® device. Featuring Robert M. Kacmarek, PhD, RRT, and David J. Pierson, MD.
Item VC32 $49.95 (nonmember $99)

To place your order, please call:

972-243-2272

Texas customers only, please add 8.25% sales tax (charged on product cost and shipping charges). Texas customers who are exempt from sales tax must supply an exemption certificate. MasterCard, Visa, Institutional Purchase Order or check payable to:

American Association for Respiratory Care

ATTN: Order Fulfillment Department
11030 Ables Lane • Dallas, Texas 75229
Phone 972-243-2272 Fax: 972-484-2720
www.aarc.org

Shipping Chart

$15 or Less	$4.25
$15.01 - $25.00	$5
$25.01 - $60.00	[illegible].25
$60.01 - $80.00	[illegible].25
$80.01 - $150.00	$10.25
$150.01 - $250.00	$14.25
$251 or more	$16.25

International Orders Add $25

News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE, New Products & Services Dept, 11030 Ables Lane, Dallas TX 75229-4593. The Reader Service Card can be found at the back of the Journal.

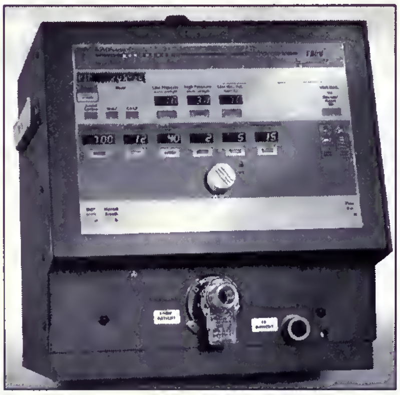
New Products & Services

Spirometer

Spacelabs Burdick Inc announces the Spirotouch, Spirometer. According to Spacelabs Burdick, this device provides a range of functions including a dynamic touchscreen, context-sensitive help screen, an automatic mode leading to user-customized testing, and automatic calibration. For more information from Spacelabs Burdick Inc, circle number 157 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

STAT Analyzer

AVL Medical Instruments announces the release of a new parameter, BUN (blood urea nitrogen), for the OMNI, Modular System. The company says the BUN measurement is performed using "thick-film technology" which eliminates the need for membrane replacements and an AVL press release says that with the addition of BUN, the OMNI is currently the only whole blood analyzer on the market that can provide the range of menu options it now offers. AVL says the other critical care parameters the analyzer measures include blood gases, electrolytes, CO-oximetery, glucose, and lactate. For more information from AVL Medical Instruments circle number 158 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/



Ventilator

Bird Products Corporation introduces the TBird Legacy ventilator. A company press release explains that this new ventilator is ideal for the home care patient, providing pressure support ventilation and flow triggering. According to Bird, these features offer the patient low work of breathing and make it very comfortable to use. The company says the Legacy ventilator employs a turbine-based system capable of operating independently of a compressed gas source. Bird says the device also provides optional external batteries for increased patient mobility. For more information from Bird Products Corporation, circle number 159 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Nasal Mask

Respironics introduces its new Profile™ Custom Nasal Mask. According to the company, this new mask allows patients to customize their mask by inserting a special inner layer of the mask into boiling water and after it cools, placing it on the face to allow the impression to set. A Respironics spokesperson said that the product provides a better fit, which in turn creates better treatment for sleep apnea and other respiratory diseases. For more information from Respironics, circle number 160 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

www.rcjournal.com

MEDWATCH

THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting
by health professionals of adverse
events and product problems

Page ____ of ____

Form Approved: OMB No. 0910-0291 Expires: 4/30/96
See OMB statement on reverse

FDA Use Only (Resp Care)

Triage unit
sequence #

A. Patient information

1. **Patient identifier**
In confidence

2. **Age at time of event:** ________
or
Date of birth:

3. **Sex**
☐ female
☐ male

4. **Weight**
____ lbs
or
____ kgs

B. Adverse event or product problem

1. ☐ **Adverse event** and/or ☐ **Product problem** (e.g., defects/malfunctions)

2. **Outcomes attributed to adverse event**
(check all that apply)
- ☐ death ____________ (mo/day/yr)
- ☐ life-threatening
- ☐ hospitalization – initial or prolonged
- ☐ disability
- ☐ congenital anomaly
- ☐ required intervention to prevent permanent impairment/damage
- ☐ other: ____________

3. **Date of event** (mo/day/yr)

4. **Date of this report** (mo/day/yr)

5. **Describe event or problem**

6. **Relevant tests/laboratory data**, including dates

7. **Other relevant history, including preexisting medical** conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. **Name** (give labeled strength & mfr/labeler, if known)
#1
#2

2. **Dose, frequency & route used**
#1
#2

3. **Therapy dates** (if unknown, give duration)
from/to (or best estimate)
#1
#2

4. **Diagnosis for use** (indication)
#1
#2

5. **Event abated after use stopped or dose reduced**
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. **Lot #** (if known)
#1
#2

7. **Exp. date** (if known)
#1
#2

8. **Event reappeared after reintroduction**
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9. **NDC #** (for product problems only)
– –

10. **Concomitant medical products** and therapy dates (exclude treatment of event)

D. Suspect medical device

1. **Brand name**

2. **Type of device**

3. **Manufacturer name & address**

4. **Operator of device**
- ☐ health professional
- ☐ lay user/patient
- ☐ other: ________

5. **Expiration date** (mo/day/yr)

6.
model # ____________
catalog # ____________
serial # ____________
lot # ____________
other #

7. **If implanted, give date** (mo/day/yr)

8. **If explanted, give date** (mo/day/yr)

9. **Device available for evaluation?** (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ____________ (mo/day/yr)

10. **Concomitant medical products** and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1. **Name & address**

phone #

2. **Health professional?**
☐ yes ☐ no

3. **Occupation**

4. **Also reported to**
- ☐ manufacturer
- ☐ user facility
- ☐ distributor

5. If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

Mail to:
MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or **FAX to:**
1-800-FDA-0178

DA Form 3500 1/96)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

• 1-800-FDA-0178	to FAX report
• 1-800-FDA-7737	to report by modem
• 1-800-FDA-1088	to report by phone or for more information
• 1-800-822-7967	for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

The public reporting burden for this collection of information has been estimated to average 30 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to:

DHHS Reports Clearance Office
Paperwork Reduction Project (0910-0291)
Hubert H. Humphrey Building, Room 531-H
200 Independence Avenue, S.W.
Washington, DC 20201

"An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number."

Please do NOT return this form to either of these addresses.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service • Food and Drug Administration

FDA Form 3500-back

Please Use Address Provided Below – Just Fold In Thirds, Tape and Mail

Department of
Health and Human Services
Public Health Service
Food and Drug Administration
Rockville, MD 20857

Official Business
Penalty for Private Use $300

NO POSTAGE
NECESSARY
IF MAILED
IN THE
UNITED STATES
OR APO/FPO

BUSINESS REPLY MAIL

FIRST CLASS MAIL PERMIT NO. 946 ROCKVILLE, MD

POSTAGE WILL BE PAID BY FOOD AND DRUG ADMINISTRATION

MEDWATCH
The FDA Medical Products Reporting Program
Food and Drug Administration
5600 Fishers Lane
Rockville, MD 20852-9787

American Association for Respiratory Care

MEMBERSHIP APPLICATION

Please read the eligibility requirements for each of the classifications in the right-hand column, then complete the applicable section. All information requested below must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

☐ Active
Associate
  ☐ Foreign
  ☐ Physician
  ☐ Industrial
☐ Special
☐ Student

Last Name ______________________________

First Name ______________________________

Social Security No. ______________________________

Home Address ______________________________

City ______________________________

State ____________ Zip ___ ___ ___ ___ ___—___ ___ ___ ___

Phone No. (______) ______________________________

Primary Job Responsibility (check one only)

☐ Technical Director
☐ Assistant Technical Director
☐ Pulmonary Function Specialist
☐ Instructor/Educator
☐ Supervisor
☐ Staff Therapist
☐ Staff Technician
☐ Rehabilitation/Home Care
☐ Medical Director
☐ Sales
☐ Student
☐ Other, specify ______________________________

Type of Business

☐ Hospital
☐ Skilled Nursing Facility
☐ DME/HME
☐ Home Health Agency
☐ Educational Institution
☐ Manufacturer or supplier
☐ Other, specify ______________________________

Date of Birth (optional) ____________ Sex (optional) ____________

U.S. Citizen? _____ Yes _____ No

Have you ever been a member of the AARC? ____________________

If so, when? From ____________________ to ____________________

Preferred mailing address: ☐ Home ☐ Business

For office use only

FOR ACTIVE MEMBER

An individual is eligible if he/she lives in the U.S. or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC. An individual who is an AARC Active Member in good standing on December 8, 1994, will continue as such provided his/her membership remains in good standing.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment ______________________________

Address ______________________________

City ______________________________

State ____________ Zip ___ ___ ___ ___ ___-___ ___ ___ ___

Phone No. (______) ______________________________

Medical Director/Medical Sponsor ______________________________

FOR ASSOCIATE OR SPECIAL MEMBER

Individuals who hold a position related to respiratory care but do not meet the requirements of Active Member shall be Associate Members. They have all the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee. The following subclasses of Associate Membership are available: Foreign, Physician, and Industrial (individuals whose primary occupation is directly or indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment or supplies). Special Members are those not working in a respiratory care-related field.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment ______________________________

Address ______________________________

City ______________________________

State ____________ Zip ___ ___ ___ ___ ___-___ ___ ___ ___

Phone No. (______) ______________________________

FOR STUDENT MEMBER

Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency.

SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member.

School/RC Program ______________________________

Address ______________________________

City ______________________________

State ____________ Zip ___ ___ ___ ___ ___-___ ___ ___ ___

Phone No. (______) ______________________________

Length of program

☐ 1 year ☐ 4 years
☐ 2 years ☐ Other, specify ____________

Expected Date of Graduation *(REQUIRED INFORMATION)*

Month ________ Year ________

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • [972] 243-2272 • Fax [972] 484-2720

American Association for Respiratory Care MEMBERSHIP APPLICATION

Demographic Questions

We request that you answer these questions in order to help us design services and programs to meet your needs.

Check the Highest Degree Earned

☐ High School
☐ RC Graduate Technician
☐ Associate Degree
☐ Bachelor's Degree
☐ Master's Degree
☐ Doctorate Degree

Number of Years in Respiratory Care

☐ 0-2 years
☐ 3-5 years
☐ 6-10 years
☐ 11-15 Years
☐ 16 years or more

Job Status

☐ Full Time
☐ Part Time

Credentials

☐ RRT
☐ CRT
☐ Physician
☐ CRNA
☐ RN
☐ LVN/LPN
☐ CPFT
☐ RPFT
☐ Perinatal/Pediatric

Salary

☐ Less than $10,000
☐ $10,001-$20,000
☐ $20,001-$30,000
☐ $30,001-$40,000
☐ $40,000 or more

PLEASE SIGN

I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics. I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

A yearly subscription to RESPIRATORY CARE journal and *AARC Times* magazine includes an allocation of $11.50 from my dues for each of these publications.

NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes. However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association lobbying activities. The AARC estimates that the nondeductible portion of your dues — the portion which is allocable to lobbying — is 26%.

Signature ____________________________________

Date ____________________________________

Membership Fees

Payment must accompany your application to the AARC. Fees are for 12 months. (NOTE: Renewal fees are $75.00 Active, Associate-Industrial or Associate-Physician, or Special status; $90.00 for Associate-Foreign status; and $45.00 for Student status).

☐ Active	$ 87.50
☐ Associate (Industrial or Physician)	$ 87.50
☐ Associate (Foreign)	$102.50
☐ Special	$ 87.50
☐ Student	$ 45.00
TOTAL	$__________

Specialty Sections

Established to recognize the specialty areas of respiratory care, these sections publish a bi-monthly newsletter that focuses on issues of specific concern to that specialty. The sections also design the specialty programming at the national AARC meetings.

☐ Adult Acute Care Section	$15.00
☐ Education Section	$20.00
☐ Perinatal-Pediatric Section	$15.00
☐ Diagnostics Section	$15.00
☐ Continuing Care-Rehabilitation Section	$15.00
☐ Management Section	$20.00
☐ Transport Section	$15.00
☐ Home Care Section	$15.00
☐ Subacute Care Section	$15.00
TOTAL	$__________

GRAND TOTAL = Membership Fee plus optional sections $__________

☐ Total Amount Enclosed/Charged $____________________
☐ Please charge my dues (see below)

To charge your dues, complete the following:
☐ MasterCard
☐ Visa

Card Number

__ __ __ __ __ __ __ __ __ __ __ __ __ __ __ __

Card Expires _______/______

Signature ____

Mail application and appropriate fees to:
American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • [972] 243-2272 • Fax [972] 484-2720

Manuscript Preparation Guide

General Information

RESPIRATORY CARE welcomes original manuscripts related to the science and technology of respiratory care and prepared according to these Instructions and the *Uniform Requirements for Manuscripts Submitted to Biomedical Journals* [Respir Care 1997; 42(6):623-634]. Manuscripts are blinded and reviewed by professionals who are experts in their fields. Authors are responsible for all aspects of the manuscript and receive galleys to proofread before publication. Each accepted manuscript is copyedited so that its message is clear and it conforms to the Journal's style. Published papers are copyrighted by Daedalus Inc and may not be published elsewhere without permission.

Editorial consultation is available at any stage of planning or writing. On request, specific guidance is provided for all publication categories. To receive these Instructions and related materials, write to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104, call (206) 223-0558, or fax (206) 223-0563.

Publication Categories & Structure

Research Article: A report of an original investigation (a study). It includes a Title Page, Abstract, Introduction, Methods, Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

Evaluation of Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification. It has a Title Page, Abstract, Introduction, Description of Device/Method/Technique, Evaluation Methods, Evaluation Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions. Comparative cost data should be included wherever possible.

Case Report: A report of a clinical case that is uncommon, or was managed in a new way, or is exceptionally instructive. All authors must be associated with the case. A case-managing physician must either be an author or furnish a letter approving the manuscript. Its components are Title Page, Abstract, Introduction, Case Summary, Discussion, References, Tables, Figures, and Figure Captions.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a pertinent topic that has been the subject of at least 40 published research articles. Title Page, Outline, Introduction, Review of the Literature, Summary, Acknowledgments, References. Tables, Appendices, and Figures and Captions may be included.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles.

Update: A report of subsequent developments in a topic that has been critically reviewed in this Journal or elsewhere.

Point-of-View Paper: A paper expressing personal but substantiated opinions on a pertinent topic. Title Page, Text, References, Tables, and Illustrations may be included.

Special Article: A pertinent paper not fitting one of the foregoing categories may be acceptable as a Special Article. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper drawing attention to a pertinent concern; it may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A signed communication, marked "For publication," about prior publications in this Journal or about other pertinent topics. Tables and illustrations may be included.

Blood Gas Corner: A brief, instructive case report involving blood gas values—with Questions, Answers, and Discussion.

Drug Capsule: A mini-review paper about a drug or class of drugs that includes discussions of pharmacology, pharmacokinetics, and pharmacotherapy.

Graphics Corner: A brief case report incorporating waveforms for monitoring or diagnosis—with Questions, Answers, and Discussion.

Kittredge's Corner: A brief description of the operation of respiratory care equipment—with information from manufacturers and editorial comments and suggestions.

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

Cardiorespiratory Interactions. A case report demonstrating the interaction between the cardiovascular and respiratory systems. It should be a patient-care scenario; however, the case—the central theme—is the systems interaction. CRI is characterized by figures, equations, and a glossary. See the March 1996 Issue of RESPIRATORY CARE for more detail.

Test Your Radiologic Skill: Like Blood Gas Corner, but involving pulmonary medicine radiography and including one or more radiographs; may involve imaging techniques other than conventional chest radiography.

Review of Book, Film, Tape, or Software: A balanced, critical review of a recent release.

Preparing the Manuscript

Print on one side of white bond paper, 8.5 in. x 11 in. (216 x 279 mm) with margins of at least 1 in. (25 mm) on all sides of the page. Use ***double-spacing*** throughout the ***entire*** manuscript. Use a standard font (eg, Times, Helvetica, or Courier) at least 10 points in size, and do not use italics except for special emphasis. Number all pages in upper-right corners. Indent paragraphs 5 spaces. Do ***not justify***. **Do *not* put authors' names, institutional affiliations or allusions to institutional affiliations in the text, or other identification anywhere except on the title page.** Repeat title only (no authors) on the abstract page. Begin each of the following on a new page: Title Page, Abstract, Text, Product Sources List, Acknowledgments, References, each Table, and each Appendix. Use standard English in the first person and active voice.

Center main section headings on the page and type them in capital and small letters (eg, Introduction, Methods, Results, Discussion). Begin subheadings at the left margin and type them in capital and small letters (eg, Patients, Equipment, Statistical Analysis).

References. Cite only published works as references. Manuscripts accepted but not yet published may be cited as references: designate the accepting journal, followed by (in press), and provide 3 copies of the in-press article for reviewer inspection. Cite references in the text with superscript numerals. Assign numbers in the order that references are first cited. On the reference page, list the cited works in numerical order. Follow the Journal's style for references. Abbreviate journal names as in *Index Medicus*. List all authors.

Article in a journal carrying pagination throughout volume:

> Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

> Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

> American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

> Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986; 89(3 Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided. Those more than 3 years old should not be cited.)

> Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in journal:

> Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

> Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

> Melony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

> Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

> DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura; 1990. p. 76-85.

Corporate author book:

> American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

Chapter in book with editor(s):

> Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott; 1977:26-42.

Tables. Use consecutively numbered tables to display information. Start each table on a separate page. Number and title the table and give each column a brief heading. Place explanations in footnotes, including all nonstandard abbreviations and symbols. Key the footnotes with conventional designations (*, †, ‡, §, ‖, ¶, **, ††) in consistent order, placing them superscript in the table body. Do not use horizontal or vertical rules or borders. Do not submit tables as photographs, reduced in size, or on oversize paper. Use the same typeface as in the text.

Illustrations. Graphs, line drawings, photographs, and radiographs are figures. Use only illustrations that clarify and augment the text. Number them consecutively as Fig. 1, Fig. 2, and so forth according to the order by which they are mentioned in the text. Be sure all figures are cited. If any figure was previously published, include copyright holder's written permission to reproduce. Figures for publication must be of professional quality. Data for the original graphs should be available to the Editor upon request. If color is essential, consult the Editor for more information. In reports of animal experiments, use schematic drawings, not photographs. A letter of consent must accompany any photograph of a person. Do not place titles and detailed explanations on figures; put this information in figure captions. If possible, submit radiographs as prints and full-size copies of film.

Drugs. Identify precisely all drugs and chemicals used, giving generic names, doses, and routes of administration. If desired, brand names may be given in parentheses after generic names. Drugs should be listed on the product-sources page.

Commercial Products. In parentheses in the text, identify any commercial product (including model number if applicable) the first time it is mentioned, giving the manufacturer's name, city, and state or country. If four or more products are mentioned, do not list any manufacturers in the text; instead, list them on a Product Sources page at the end of the text, before the References. Provide model numbers when available and manufacturer's suggested price, if the study has cost implications.

Ethics. When reporting experiments on human subjects, indicate that procedures were conducted in accordance with the ethical standards of the *World Medical Association Declaration of Helsinki* [Respir Care 1997;42(6):635-636] or of the institution's committee on human experimentation. State that informed consent was obtained. Do not use patient's names, initials, or hospital numbers in text or illustrations. When reporting experiments on animals, indicate that the institution's policy, a national guideline, or a law on the care and use of laboratory animals was followed.

Statistics. Identify the statistical tests used in analyzing the data, and give the prospectively determined level of significance in the Methods section. Report actual p values in Results. Cite only textbook and published article references to support choices of tests. Identify any general-use or commercial computer programs used, naming manufacturers and their locations. These should be listed on the product-sources page.

Units of Measurement. Express measurements of length, height, weight, and volume in metric units appropriately abbreviated; temperatures in degrees Celsius; and blood pressures in millimeters of mercury (mm Hg). Report hematologic and clinical-chemistry measurements in conventional metric and in SI (*Systémé Internationale*) units. Show gas pressures (including blood gas tensions) in torr. List SI equivalent values, when possible, in brackets following non-SI values—for example, "PEEP, 10 cm H_2O [0.981 kPa]." For conversion to SI, see RESPIRATORY CARE 1988;33(10):861-873 (Oct 1988), 1989;34(2):145 (Feb 1989), and 1997;42(6):639-640 (June 1997).

Conflict of Interest. Authors are asked to disclose any liaison or financial arrangement they have with a manufacturer or distributor whose product is part of the submitted manuscript or with the manufacturer or distributor of a competing product. (Such arrangements do not disqualify a paper from consideration and are not disclosed to reviewers.) A statement to this effect is included on the cover-letter page. (Reviewers are screened for possible conflict of interest.)

Abbreviations and Symbols. Use standard abbreviations and symbols. Avoid creating new abbreviations. Avoid all abbreviations in the title and unusual abbreviations in the abstract. Use an abbreviation only if the term occurs several times in the paper. Write out the full term the first time it appears, followed by the abbreviation in parentheses. Thereafter, employ the abbreviation alone. Never use an abbreviation without defining it. Standard units of measurement can be abbreviated without explanation (eg, 10 L/min, 15 torr, 2.3 kPa).

Please use the following forms: cm H_2O (not cmH2O), f (not bpm), L (not l), L/min (not LPM, l/min, or lpm), mL (not ml), mm Hg (not mmHg), pH (not Ph or PH), $p > 0.001$ (not p>0.001), s (not sec), S_{pO_2} (pulse-oximetry saturation). See RESPIRATORY CARE: Standard Abbreviations and Symbols [Respir Care 1997;42(6):637-642].

Submitting the Manuscript

Mail three copies [1 copy with author(s) name(s), affiliation(s), 2 copies without name(s) and affiliation(s) for reviewers] of the manuscript, figures, and 1 diskette, and the Cover Letter & Checklist to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104. Do not fax manuscripts. Protect figures with cardboard. Keep a copy of the manuscript and figures. Receipt of your manuscript will be acknowledged.

Computer Diskettes. Authors are encouraged to submit electronic versions of manuscripts as well as printed copies (3.5 in. diskettes in Macintosh or IBM-DOS format). Label each diskette with date; author's name; name and version of word-processing program used; and filename(s). Software used to produce graphics and tables should be similarly identified. Do not write on diskette labels except with felt-tipped pen. If revision of a manuscript is required as a condition of acceptance for publication, we ask that an electronic version of revision be supplied to facilitate copyediting and production.

Prior and Duplicate Publication. Work that has been published or accepted elsewhere should not be submitted. In special instances, the Editor may consider such material, provided that permission to publish is given by the author and original publisher. Please consult the Editor before submitting such work.

Authorship. All persons listed as authors should have participated in the reported work and in the shaping of the manuscript; all must have proofread the submitted manuscript; and all should be able to publicly discuss and defend the paper's content. A paper with corporate authorship must specify the key persons responsible for the article. Authorship is not justified solely on the basis of solicitation of funding, collection or analysis of data, provision of advice, or similar services. Persons who provide such ancillary services exclusively may be recognized in an Acknowledgments section.

Permissions. The manuscript must be accompanied by copies of permissions to reproduce previously published material (figures or tables); to use illustrations of, or report sensitive personal information about, identifiable persons; and to name persons in the Acknowledgments section.

Reviewers. Please supply the names, credentials, affiliations, addresses, and phone/fax numbers of three professionals whom you consider expert on the topic of your paper. Your manuscript may be sent to one or more of them for blind peer review.

Editorial Office:

RESPIRATORY CARE
600 Ninth Avenue, Suite 702
Seattle WA 98104

(206) 223-0558 (voice)
(206) 223-0563 (fax)
e-mail: rcjournal@aarc.org
kreilkamp@aarc.org

COVER LETTER & CHECKLIST

A copy of this completed form must accompany all manuscripts submitted for publication.

Title of Paper: ____________________

Publication Category: ____________________

Corresponding Author: ____________________ Phone: __________ FAX: __________

Mailing Address: ____________________

Reprints: ☐ Yes ☐ No E-mail Address: ____________________

"We, the undersigned, have all participated in the work reported, proofread the accompanying manuscript, and approve its submission for publication." Please print and include credentials, title, institution, academic appointments, city and state. If more than 4 authors, please use another copy of this form.*

*First Author: ____________________

Author Signature/Date ____________________

*Second Author: ____________________

Author Signature/Date ____________________

*Third Author: ____________________

Author Signature/Date ____________________

*Fourth Author: ____________________

Author Signature/Date ____________________

Has this research been presented in any public forum? ☐ Yes ☐ No

If yes, where, when and by whom? ____________________

Has this research received any awards? ☐ Yes ☐ No

If yes, please describe. ____________________

Has this research received any grants or other support, financial or material? ☐ Yes ☐ No

If yes, please describe. ____________________

Do any of the authors of this manuscript have a financial interest in (or a commercial or consulting relationship to) any of the products or manufacturers mentioned in this paper or any competing products or manufacturers? ☐ Yes ☐ No

If yes, please describe. ____________________

CHECKLIST

- ☐ Have you enclosed a copy of the manuscript on diskette?
- ☐ Is double-spacing used throughout entire manuscript?
- ☐ Are all pages numbered in upper-right corners?
- ☐ Are all references, figures, and tables cited in the text?
- ☐ Has the accuracy of the references been checked, and are they correctly formatted?
- ☐ Have SI values been provided?
- ☐ Has all arithmetic been checked?
- ☐ Have generic names of drugs been provided?
- ☐ Have necessary written permissions been provided?
- ☐ Have authors' names been omitted from text and figure labels?
- ☐ Have copies of 'in press' references been provided?
- ☐ Has the manuscript been proofread by all the authors?
- ☐ Have the manufacturers and their locations been provided for all devices and equipment used?

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of the month two months preceding the month in which you wish the ad to run. Submit copy and insertion orders to Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

Calendar of Events

AARC & AFFILIATES

August 25–27—*Cleveland, Ohio*
The OSRC state conference will be held at the Holiday Inn in Independence, just south of Cleveland. Specialty sessions include critical care, pediatrics, rehab/ continuing care, research, and management.
Contact: Joe Huff at (216) 861-6200, ext. 3892, **ewlu18a@prodigy.com** or Nancy Johnson at (330) 929-7166, **abbyru@aol.com**.

September 8-10—*Hot Springs, Arkansas*
The ASRC presernts its 28th Annual State Meeting and Education Seminar at the Hot Springs Hilton and Convention Center. Topics will include health care in Arkansas, patient rights, disease management, and case studies by area RTs. Scheduled to speak are State Representative Brenda Gullett, Congressman Vic Snyder, Tom Kallstrom of Cleveland, OH, ASRC President John Campbell, AARC President Dianne Kimball, and Kristin McFall of Denver, CO (topic: cystic fibrosis).
Contact: John Lindsey at (870) 541-7606, jlindsey@ahecpb.uams.edu

September 16–17—*Pittsburgh, Pennsylvania*
The PSRC will host their 26th Annual Western Pennsylvania Regional Pulmonary Medicine and Physiology Conference at the Sheraton Station Square. Topics include management, critical care, sleep diagnostics, pulmonary rehabilitation, and the physician forum.
Contact: Debbie Logan at (800) 545-4663, ext. 112.

September 24–25—*Cleveland, Ohio*
The AARC presents the "Disease Management of Asthma" seminar. Come and join a distinguished faculty as they review the NIH asthma guidelines, marketing the asthma program, pharmacology, and numerous other aspects of asthma program management.
Contact: The AARC Conventions Office at (972) 243-2272.

October 1—*Melville, New York*
The NYSSRC's Southeastern Chapter hosts their 31st annual symposium, "Respiratory Care — A Work in Progress," at the Huntington Hilton Hotel in Melville, Long Island. The keynote address will be given by Carl Wiezalis, vice-president of the AARC.
Contact: For information, call Jim Ganetis at (516) 444-3181 or **www.nyssrc.org**.

October 20–22—*Daniels, West Virginia*
The West Virginia Society for Respiratory Care will host its Annual Fall Meeting at the Glade Springs Resort, Country Inns and Suites.
Contact: For more information, contact Jay Wildt, co-chair of program and education, at (304) 442-7474.

December 13–16—*Las Vegas, Nevada*
The AARC's 45th International Respiratory Congress is scheduled for Dec. 13–16 (Monday through Thursday) at the Las Vegas Convention Center. Sessions appealing to all levels of health care providers will be offered, with CRCE credit available. Exhibits by international manufacturers of cardiopulmonary equipment will be featured. Additional information will be available in the fall.

Other Meetings

August 19–20—*Cleveland, Ohio*
The Cleveland Clinic Foundation is sponsoring a continuing education program titled "Respiratory Therapy," which has been approved for Category 1 accreditation. It will be held at the Omni International Hotel.
Contact: For more information, call Laurie Martel at (216) 444-5696 or (800) 862-8173.

September 16-19—*Phoenix, Arizona*
The American Association of Cardiovascular and Pulmonary Rehabilitation will hold its 14th annual meeting at the Phoenix Civic Center.
Contact: For more information, call (608) 831-6989; aacvpr@tmahq.com; http://www.aacvpr.org.

October 1–3—*Ottawa, Ontario, Canada*
The Canadian COPD Alliance will host "Building and Enriching Partnerships in the Management of COPD" at the Radisson Hotel Ottawa Centre. This conference will include plenary sessions on the epidemiology of COPD and scientific workshops on spirometry, smoking cessation, and the evidence to support management approaches to COPD. Included is a series of practical workshops on rehabilitation and a parallel consumer track for those who live with COPD.
Contact: call (613) 747-6776 or see their web site at **www.lung.ca/CCA/ conference**.

October 4–5—*Ann Arbor, Michigan*
The Office of Continuing Medical Education at the University of Michigan is sponsoring a conference, "Update on Pulmonary and Critical Care Medicine," at the Towsley Center.
Contact: For more information, contact Laura Castellanos at (734) 647-8784.

October 31–November 4—*Chicago, Illinois*
The American College of Chest Physicians will host their 65th Annual International Scientific Assembly at the Lakeside Center. For information, contact Member Services at (800) 343-2227, fax (847) 498-5460, or **www.chestnet.org**.

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

Las Vegas

45TH INTERNATIONAL RESPIRATORY CONGRESS
DECEMBER 13–16, 1999
LAS VEGAS, NEVADA

1999

Helpful Web Sites

American Association for Respiratory Care
http://www.aarc.org
- —Current job listings
- —American Respiratory Care Foundation fellowships, grants, & awards
- —Clinical Practice Guidelines

National Board for Respiratory Care
http://www.nbrc.org

RESPIRATORY CARE online
http://www.rcjournal.com
- —1997 Subject and Author Indexes
- —Contact the editorial staff

Asthma Management Model System
http://www.nhlbi.nih.gov

The National Board for Respiratory Care—1999 Examination Dates and Fees

Examination	Examination Date	Examination Fee
CRTT (CRT) Examination	November 13, 1999 Application Deadline: September 1, 1999	$120 (new applicant) 80 (reapplicant)

For information about other services or fees, write to the National Board for Respiratory Care, 8310 Nieman Road, Lenexa KS 66214, or call (913) 599-4200, FAX (913) 541-0156,or e-mail: nbrc-info@nbrc.org

WATCH FOR
SPECIAL ISSUE

THORACIC
IMAGING IN
THE INTENSIVE
CARE UNIT

SEPTEMBER 1999

Asthma Disease Management Course Comes to Cleveland

"Disease Management of Asthma," a newly designed day-and-a-half course, will be held in Cleveland Sept 24-25. Drawing on the expertise of a nationally recognized faculty, this course has been developed in accordance with the National Institute of Health *Asthma Guidelines for the Diagnosis and Management of Asthma*. Completing this course will earn respiratory therapists 11 hours of continuing education credit. Topics covered include disease management, pharmacology, educational strategies, marketing your program, outcomes management, and age-specific issues. Register on-line or call the AARC for registration materials. The program will be held September 24-25, at the Marriott Cleveland Airport Hotel. Registration before August 30, 1999 is $175 for AARC members, $250 for non-members. (After August 30, register for $275 member, $350 non-member)

AARC Exhibition Becomes 'Selling Show'

The AARC's 45th International Respiratory Congress will, for the first time in its history, provide attendees and exhibitors the opportunity to negotiate sales of products and equipment at the exhibitor show, December 13 -16, in Las Vegas. More than 200 equipment manufacturers, pharmaceutical companies, service organizations, and supply companies will exhibit at the show. "We've always had an extremely enthusiastic response to our exhibiting companies," said AARC Executive Director Sam Giordano. "We decided to take that approach this year to make attending the meeting even more cost effective — and the show specials and discounts our exhibitors are providing will make the exhibition an even greater success," he said.

New Products Help with Clinical Practice

These products from the AARC, some newly introduced, will help in both clinical practice situations and in administration, in acute care and post-acute care. Check them out:

Diagnostic Training and Competence Assessment Manual for Pulmonary and Noninvasive Cardiology. CDRom. $267 for AARC members. This manual (on compact disk) is ideal for use in course development, training, orientation, and competence assessment for individuals in the laboratory setting. A pulmonary diagnostics section features quality control, diffusing capacity, whole body plethysmography, arterial blood gas sampling, bronchoscopy and seven other procedures. The noninvasive cardiology section features EEG, stress testing, event monitoring, exercise testing and three other procedures.

Asthma Disease State Management. Video and workbook. $79.95 for members. Provides instruction in how to create an effective asthma disease management program in your facility. Covers diagnosis, pharmacological therapy, environmental controls, patient/family education, and case studies. Approved for two hours of CRCE credit and nursing CE credit.

Orientation and Competency Assurance Documentation Manual. Manual. $65 for members. Provides the information, assessment tools, and models necessary to demonstrate that the competence of employees is documented according to JCAHO requirements.

Uniform Reporting Manual for Acute Care. Manual. $65 for members. Provides you with nationally recognized standards for documenting workload units and time standards. Includes patient assessment activities and covers bronchial hygiene, supplemental oxygen, airway care, diagnostic tests, and cardiovascular diagnostics.

Uniform Reporting Manual for Subacute Care. Manual. $75 for members. Provides tools to determine productivity, track trends in the utilization of respiratory care services, assist in determining personnel requirements, measure demand for and intensity of services, and meet the requirements of prospective payment systems (PPS).

Respiratory Home Care Procedure Manual. Manual. $80 for members. The new "Respiratory Home Care Procedure Manual" is specifically designed for the home care setting. And, it is easily adaptable to any alternate care site from subacute to home medical equipment companies and nursing agencies. The manual features five sections of information, forms, and checklists for the patient and practitioner.

Authors in This Issue

Adams, Alexander B	918, 925
Ahmad, Muzaffar	959
Bliss, Peter L	918, 925
Branson, Richard D	912
Bugedo, Guillermo	918
Campbell, Robert S	912
Davis, Kenneth Jr	912
Demers, Bob	963
Dhand, Rajiv	940
Durbin, Charles G Jr	909
Fink, James	940
Fluck, Robert R Jr	965
Frame, Scott	912
Grissom, Colin K	968
Helmholz, H Frederic Jr	952, 965
Hunton, Deborah A	955
Johannigman, Jay A	912
Kaminsky, David A	955
Kasper, Carol L	968
Kathawalla, Salim	959
Knebel, Ann R	932
Leidy, Nancy Kline	932
Lewis, Robert	967
Luchette, Fred A	912
Marini, John J	918
McCoy, Robert W	925
Morfei, Joe	963
Ottaway, Michael	912
Schilz, Robert J	961
Takahashi, Toshimichi	918

Advertisers in This Issue

To advertise in RESPIRATORY CARE, contact Tim Goldsbury, 20 Tradewinds Circle, Tequesta FL 33469 at (561) 745-6793, Fax (561) 745-6795, e-mail: goldsbury@aarc.org, for rates and media kits. For recruitment/classified advertising contact Beth Binkley, Marketing Assistant for RESPIRATORY CARE, at (972) 243-2272, Fax (972) 484-6010. Rick Owen is the Marketing Director for RESPIRATORY CARE.

AirSep Corporation	889	Circle Reader Service No. 140	Call (800) 874-0202
Datex-Ohmeda	905	Circle Reader Service No. 129	Call (800) 345-2700
DEY Laboratories	881-882	Circle Reader Service No. 114	Call (800) 755-5560
DEY Laboratories	Cover 2	Circle Reader Service No. 133	Call (800) 755-5560
DHD Healthcare	Cover 4	Circle Reader Service No. 110	Call (800) 847-8000
Hamilton Medical, Inc.	897	Circle Reader Service No. 119	Call (800) HAM-MED-1
Hans Rudolph	893	Circle Reader Service No. 120	Call (800) 456-6695
IngMar Medical	891	Circle Reader Service No. 124	Call (800) 583-9910
Masimo Corporation	884	Circle Reader Service No. 106	Call (800) 4-MASIMO
Maxtec, Inc.	895	Circle Reader Service No. 117	Call (800) 748-5355
Sims-Portex, Inc.	Cover 3	Circle Reader Service No. 130	Call (800) 258-5361
Vortran Medical Technology 1, Inc.	903	Circle Reader Service No. 112	Call (800) 434-4034
Westmed, Inc.	886	Circle Reader Service No. 104	Call (800) 724-2328

COPYRIGHT INFORMATION. RESPIRATORY CARE is copyrighted by Daedalus Enterprises Inc. Reproduction in whole or in part without the express written permission of Daedalus Enterprises Inc is prohibited. Permission to photocopy a single article in this Journal for noncommercial purposes of scientific or educational advancement is granted. Permission for multiple photocopies and copies for commercial purposes must be requested in writing, via e-mail (rcjournal@aarc.org), or telephone and approved by RESPIRATORY CARE. Anyone may, without permission, quote up to 500 words of material in this journal provided the quotation is for noncommercial use and RESPIRATORY CARE is credited. Longer quotation requires written approval by the author and publisher. Single reprints are available only from the authors. Reprints for commercial use may be purchased from Daedalus Enterprises Inc. For more information and prices call (972) 243-2272.

DISCLAIMER. The opinions expressed in any article or editorial are those of the author and do not necessarily reflect the views of the Editors, the American Association for Respiratory Care (AARC), or Daedalus Enterprises Inc. Neither are the Editors, the AARC, or the Publisher responsible for the consequences of the clinical applications or use of any methods or devices described in any article or advertisement.

SUBSCRIPTION RATES. Individual subscription rates are $75 per year (12 issues), $145 for 2 years, and $215 for 3 years in the US and Puerto Rico. Rates are $90 per year, $175 for 2 years, and $260 for 3 years in all other countries (add $94 per year for air mail). Single copies when available cost $7; add $9 for air mail postage to overseas countries. Checks should be made payable to RESPIRATORY CARE and sent to the subscription office at 11030 Ables Lane, Dallas TX 75229-4593.

SUBSCRIPTION RATES FOR ASSOCIATIONS. Basic annual subscriptions are offered to members of associations according to their membership enrollment: 101-500 members = $13.50/year; 501-1,500 = $13/year; 1,500-10,000 = $12.50/year; more than 10,000 = $11.50/year. Individual subscriptions ar available at these rates: $75/year (12 issues in the United States or Puerto Rico; $90/year in other countries. For information, contact Ray Masferrer at (972) 243-2272.

CHANGE OF ADDRESS. Notify the AARC at (972) 243-2272 as soon as possible of any change in address. Note the subscription number (from the mailing label) and your name, old address, and new address. Allow 6 weeks for the change. To avoid charges for replacement copies of missed issues, requests must be made within 60 days in the US and 90 days in other countries.

MANUSCRIPTS. The Journal publishes clinical studies, method/device evaluations, reviews, and other materials related to cardiopulmonary medicine and research. Manuscripts may be submitted to the Editorial Office, RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104. Instructions for authors are printed in every issue. An expanded version of the Instructions is available from the editorial office.

Copyright © 1999, by Daedalus Enterprises Inc.

RESPIRATORY CARE

We are pleased to offer ***CRCE through the Journal*** for 1999. On the other side of this page, you will find space for your name and mailing address and a grid for your AARC member number and a grid for answering the questions. Complete the grids by using these rules:

Only original Answer Sheets will be graded. (This page is perforated to make it easy to complete and mail.) No facsimiles or photocopies will be accepted. Six hours of CRCE credit will be awarded to those who answer 35 or more questions correctly.

You may photocopy the Answer Sheet for your personal record. The Answer Key will be published in the October issue of RESPIRATORY CARE.

Obtain a check or money order for $10 payable to AARC. (This is for processing; your Answer Sheet will not be graded if the fee is not included.)

Read and sign the statement below.

Place the Original Answer Sheet and processing fee in a stamped #10 envelope and mail to:

CRCE through the Journal
11030 Ables Lane
Dallas TX 75229-4593

Deadline is September 30, 1999. Responses postmarked after September 30, 1999 will not be processed.

Follow all instructions explicitly. Failure to do so may cause technical problems that could disqualify you.

Questions concerning the examination should be directed to the CRCE Coordinator, (972) 243-2272.

Remember, the acceptance of these credits for the fulfillment of license-mandated continuing education is dictated solely by the licensure law of each individual state.

I attest that I completed the Answer Sheet independently, without help from others.

Signature

Date

CRCE through the Journal — Answer Sheet

See the bottom of this page for instructions on marking this form.

	a	b	c	d	e
1	○	○	○	○	○
2	○	○	○	○	○
3	○	○	○	○	○
4	○	○	○	○	○
5	○	○	○	○	○
6	○	○	○	○	○
7	○	○	○	○	○
8	○	○	○	○	○
9	○	○	○	○	○
10	○	○	○	○	○
11	○	○	○	○	○
12	○	○	○	○	○
13	○	○	○	○	○
14	○	○	○	○	○
15	○	○	○	○	○
16	○	○	○	○	○
17	○	○	○	○	○
18	○	○	○	○	○
19	○	○	○	○	○
20	○	○	○	○	○
21	○	○	○	○	○
22	○	○	○	○	○
23	○	○	○	○	○
24	○	○	○	○	○
25	○	○	○	○	○
26	○	○	○	○	○
27	○	○	○	○	○
28	○	○	○	○	○
29	○	○	○	○	○
30	○	○	○	○	○
31	○	○	○	○	○
32	○	○	○	○	○
33	○	○	○	○	○
34	○	○	○	○	○
35	○	○	○	○	○
36	○	○	○	○	○
37	○	○	○	○	○
38	○	○	○	○	○
39	○	○	○	○	○
40	○	○	○	○	○
41	○	○	○	○	○
42	○	○	○	○	○
43	○	○	○	○	○
44	○	○	○	○	○
45	○	○	○	○	○
46	○	○	○	○	○
47	○	○	○	○	○
48	○	○	○	○	○
49	○	○	○	○	○
50	○	○	○	○	○

Please print your name and AARC member number exactly as it appears on the mailing label and carefully fill in the corresponding circles below.

Member Name:

LAST ____________ FIRST ____________ MI ____

Mailing Address:

STREET ____________

CITY ____________ STATE ____________ ZIP ____________

Telephone Numbers:

HOME ____________

BUSINESS ____________

AARC Member Number:

	_	_	_	_	_	_	_
0	○	○	○	○	○	○	○
1	○	○	○	○	○	○	○
2	○	○	○	○	○	○	○
3	○	○	○	○	○	○	○
4	○	○	○	○	○	○	○
5	○	○	○	○	○	○	○
6	○	○	○	○	○	○	○
7	○	○	○	○	○	○	○
8	○	○	○	○	○	○	○
9	○	○	○	○	○	○	○

For office use only

Total correct ☐

Percent correct ☐

☐ Pass ☐ Fail

Second Fold Here

First Fold Here

Tips to ensure accurate grading and proper credit:

- Mark only **one answer**.
- If you must change your answer, **erase completely**.
- Make dark marks and **fill the circle** completely.

Examples

Do make your marks like this: ○ ○ ● ○ ○

Do Not make marks like this: ○ ✓ ○ ○ ○

Do Not make marks like this: ○ ○ ○ ☒ ○

Portex® Arterial Blood Sampling Devices

Needle-Pro® Needle Protection Devices for Maximum Needle Protection for Clinicians

Filter-Pro® Devices for Easy Removal of Air Bubbles

Pro-Vent® Plus Microsampling Devices for Small, Fixed Volume Blood Samples

Complete Family of Line Draw Syringes for Direct Blood Sampling from an Arterial Line

©1[illegible] SIMS Portex Inc.

Subscription

Respiratory Care Subscription Form

Respiratory Care is the most highly regarded peer-reviewed scientific publication for the Clinician participating in the evaluation and care of patients with cardiopulmonary problems. Please print; incomplete forms will not be processed. For faster service, fax to (972)484-2720

DON'T MISS A SINGLE ISSUE!

	U.S. Subscriptions	Subscriptions Outside U.S.
1 YR (12 issues)	☐ $75	☐ $90
2 YRS (24 issues)	☐ $145	☐ $175
3 YRS (36 issues)	☐ $215	☐ $260

Enclosed is a check in the amount of
$ ____________

Charge to my:
- ☐ Mastercard
- ☐ Visa
- ☐ Bill Me

Signature ____ Expiration Date ____ Credit Card Number ____

Name ____ Title ____

Facility Name ____

Address ____

City ____ State ____ Zip Code ____ Country ____

Telephone ____ Fax ____

RESPIRATORY CARE

FREE

Product Information

To receive information on the products and services mentioned in this issue, circle the corresponding category number or individual manufacturer's number. Fill in your name and address and mail this postage-paid card. Information will be sent directly to you from the manufacturer. Please print; incomplete forms will not be processed. For faster service, fax to (609)786-4415

MAIL TODAY!

For AARC membership information, circle 101. For Respiratory Care subscription information, circle 102.

101	102	103	104	105	106	107	108	109	110	111	112	113	114	115	116	117	118
119	120	121	122	123	124	125	126	127	128	129	130	131	132	133	134	135	136
137	138	139	140	141	142	143	144	145	146	147	148	149	150	151	152	153	154
155	156	157	158	159	160	161	162	163	164	165	166	167	168	169	170	171	172
173	174	175	176	177	178	179	180	181	182	183	184	185	186	187	188	189	190
191	192	193	194	195	196	197	198	199	200								

Please circle no more than 15 items.

Name ____ Title ____

Facility Name ____

Address ____

City ____ State ____ Zip Code ____ Country ____

Telephone ____ Fax ____

I TYPE OF INSTITUTION OR PRACTICE
- 1 ☐ Hospital
- 2 ☐ Skilled Nursing Facility
- 3 ☐ Subacute Care Facility
- 4 ☐ Home Care Practice
- 5 ☐ School
- 6 ☐ Distributor

II DEPARTMENT
- A ☐ Respiratory Care
- B ☐ Cardiopulmonary
- C ☐ Subacute Care
- D ☐ Home Care

III SPECIALTY
- 1 ☐ Clinician
- 2 ☐ Perinatal/ Pediatrics
- 3 ☐ Critical Care
- 4 ☐ Research
- 5 ☐ Subacute Care
- 6 ☐ Diagnostics/ Pulmonary Function
- 7 ☐ Management
- 8 ☐ Home Care
- 9 ☐ Rehabilitation
- 10 ☐ Education

IV POSITION
- A ☐ Department Head
- B ☐ Chief Therapist
- C ☐ Supervisor
- D ☐ Staff Therapist/ Technician
- E ☐ Medical Director
- F ☐ Educator/Instructor
- G ☐ Sales
- H ☐ Other (please specify) ____

V ARE YOU AN AARC MEMBER?
1 ☐ Yes 2 ☐ No

CRCE through the Journal — Answer Sheet

See the bottom of this page for instructions on marking this form.

Please print your name and AARC member number exactly as it appears on the mailing label and carefully fill in the corresponding circles below.

Member Name:

____________________ LAST ____________________ FIRST ____ MI

	a	b	c	d	e
1	○	○	○	○	○
2	○	○	○	○	○
3	○	○	○	○	○
4	○	○	○	○	○
5	○	○	○	○	○
6	○	○	○	○	○
7	○	○	○	○	○
8	○	○	○	○	○
9	○	○	○	○	○
10	○	○	○	○	○
11	○	○	○	○	○
12	○	○	○	○	○
13	○	○	○	○	○
14	○	○	○	○	○
15	○	○	○	○	
16	○	○	○	○	
17	○	○	○	○	
18	○	○	○	○	
19	○	○	○	○	
20	○	○	○	○	
21	○	○	○	○	
22	○	○	○	○	
23	○	○	○	○	
24	○	○	○	○	
25	○	○	○	○	
26	○	○	○	○	
27	○	○	○	○	
28	○	○	○	○	
29	○	○	○	○	
30	○	○	○	○	
31	○	○	○	○	
32	○	○	○	○	
33	○	○	○	○	
34	○	○	○	○	
35	○	○	○	○	
36	○	○	○	○	
37	○	○	○	○	
38	○	○	○	○	
39	○	○	○	○	
40	○	○	○	○	
41	○	○	○	○	
42	○	○	○	○	
43	○	○	○	○	
44	○	○	○	○	
45	○	○	○	○	
46	○	○	○	○	
47	○	○	○	○	
48	○	○	○	○	
49	○	○	○	○	
50	○	○	○	○	

Tips to ensure accurate

- Mark only **one an**
- If you must chang
- Make dark marks

NO POSTAGE
NECESSARY
IF MAILED
IN THE
UNITED STATES

BUSINESS REPLY MAIL

FIRST-CLASS MAIL PERMIT NO. 2480 Dallas,TX

POSTAGE WILL BE PAID BY ADDRESSEE

AARC Subscriptions
ATTN: Beth Binkley
PO BOX 29686
Dallas TX 75229-9691

NO POSTAGE
NECESSARY
IF MAILED
IN THE
UNITED STATES

BUSINESS REPLY MAIL

FIRST-CLASS MAIL PERMIT NO. 881 Riverton, NJ

POSTAGE WILL BE PAID BY ADDRESSEE

AARC Publications
PO BOX 11605
Riverton NJ 08076-7205

Portex® Arterial Blood Sampling Devices

When the chemistry is right, the possibilities are endless.

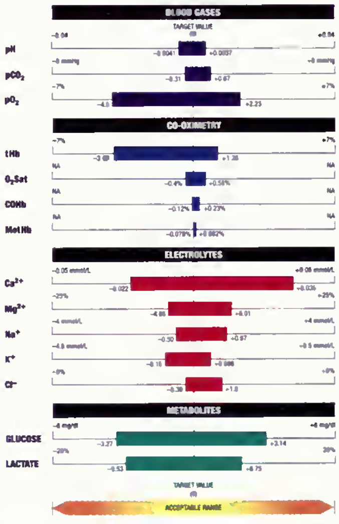
The right chemistry can spark great relationships, inspire genius, and produce medical breakthroughs. Case in point: SIMS Portex advanced formula heparin makes arterial blood sampling easier, more accurate, and more efficient, permitting 11 tests from a single sample. With such inspired innovations, the relationship between our ABS products and discriminating clinicians will go on forever.

Our chemistry is so right that one sample is all you need for 11 tests.

Our advanced formula heparin combines the necessary anticoagulant effect with calcium-neutral heparin for more accurate, more extensive testing from a single sample. Ours is the first and only heparin formula that can measure 11 indications from one arterial blood sample.

Endless possibilities, more products, lots of safety features.

SIMS Portex offers more product choices than any other manufacturer—liquid and dry heparin, with syringe sizes ranging from 250 µL to 3 cc.



Data based on independent studies whose population was composed of ICU and ER patients.

We also help to eliminate the frightening possibility of needlesticks with our patented Needle-Pro® needle protection device; the Filter-Pro® air bubble removal device is standard on all our syringes.

That special spark?

There is a special chemistry between SIMS Portex and the clinicians who use our products, a relationship built on trust, support, and reliability. Clinicians prefer our blood sampling devices for obvious reasons: they are safer, easier to use, and less traumatic for patients.

Newly released on videotape.

Call us, toll-free, to get your tape of our newest video release on arterial blood sampling devices and techniques. Ask for Nicole Hall, 1-800-258-5361, prompt 4, ext 226.

For further information, contact:

PORTEX

SIMS Portex Inc.

800-258-5361 or Fax 603-352-3703
www.portexusa.com

SIMS SMITHS INDUSTRIES
Medical Systems

Portex®, Pro-Vent®, Needle-Pro®, and Filter-Pro® are registered trademarks, and SIMS™ is a trademark of Smiths Industries Medical Systems. Point-Lok™ is a trademark of Noved Medical.

Circle 130 on reader service card

Needle-Pro® Needle Protection Devices for Maximum Needle Protection for Clinicians

Filter-Pro® Devices for Easy Removal of Air Bubbles

Pro-Vent® Plus Microsampling Devices for Small, Fixed Volume Blood Samples

Complete Family of Line Draw Syringes for Direct Blood Sampling from an Arterial Line

©1998 SIMS Portex Inc.

The new RFb System-1.™

Reduce patient anxiety during respiratory retraining.

Now you can help respiratory patients relearn controlled (diaphragmatic) breathing by helping them forget all about it. The RFb System-1 is a breakthrough biofeedback tool that significantly reduces patient anxiety through visualization of diaphragmatic breathing. The result is therapy that's easier—and less stressful—on *both* of you.

A noninvasive sensor reflects diaphragmatic movement back to the brain through an audio/visual headset (patient can choose between white noise and a musical tone). The patient receives continuous reinforcement of the correct deep-breathing technique without electrodes or other invasive devices. A computer console makes it easy to monitor patient progress with minimal therapist involvement. Therapy has been reimbursed under Medicare B. For more information, call DHD Healthcare toll-free today: **1-800-847-8000.**

DHD Healthcare
Innovations for respiratory care
DHD Healthcare Corporation

AARC ZENITH AWARD '96,'97 & '98

125 Rasbach Street Canastota, NY 13032 USA • (315) 697-2221• FAX: (315) 697-5191 • www.dhd.com

RFb System-1 is a trademark of DHD Healthcare Corporation.

© 1999 DHD Healthcare Corporation

Circle 110 on reader service card